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SYMPOSIUM ON SINUS THROMBOSIS.

I.—AN OBSCURE CASE OF SINUS THROMBOSIS WITH UNUSUAL COMPLICATIONS.*

DR. MANFRED J. GERSTLEY, New York City.

Due to limited time I shall only touch upon the poignant facts of the case I am presenting.

A white male, age 13 years, was admitted to the service of Dr. Marvin F. Jones at the New York Post-Graduate Hospital on May 18, 1933. He complained of partial loss of hearing, bilateral earache, a discharging left ear and fever.

One week prior to his admission, and following a cold, he developed a pain in the left ear which began discharging spontaneously four days later. The right ear at the same time began losing some of its acuity. A few days later a mild hyperesthesia developed over his entire body with a marked tenderness of his right thigh.

On admission the patient's temperature was 104°. In general appearance he was well nourished, inclined to obesity and slightly flushed, but with no indications of intense suffering.

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The mucous membranes of the nose and throat were in a state of acute congestion with a moderate nasal and post-nasal discharge and subacute tonsillitis.

The right ear showed no abnormalities of the canal. The drum, though intact, was thickened, showed a loss of normal luster and in general appeared as a resolving O. M. P. A. with tenderness over the antrum.

The left ear also presented the appearance of an O. M. P. A. with tenderness over the mastoid antrum. The canal was filled with a thick purulent pulsating discharge emerging through a large perforation in the inferoanterior quadrant.

As mentioned above, the extreme sensitivity of the right thigh necessitated a consultation, which gave us negative findings. Due to the septic temperature curve we arrived at a diagnosis of acute mastoiditis with possible involvement of the sigmoid sinus.

A transfusion was ordered and the blood cultured. A positive report was returned, confirming our suspicions. Streptococcus hemolyticus was the organism found.

I will not go into detail concerning the various laboratory examinations, but nothing having any bearing on the case was omitted.

Shortly thereafter the left mastoid was exenterated and the internal jugular vein and sigmoid sinus obliterated. Streptococcus hemolyticus was also found in the culture of mastoid pus and jugular vein thrombus.

The temperature after a temporary drop resumed a septic character with practically no improvement after repeated transfusions (seven being given within a month, ranging in quantity from 250 to 500 cc. each).

Repeated blood cultures proved negative after the operation and further medical and surgical consultants could find no demonstrable pathology causing his abnormal temperature.

During this time the right leg was kept in flexion continually and because of the extreme sensitivity a metal basket was used for protection from the bed clothes.

After much deliberation the right mastoid was opened four weeks later, although the canal, drum and Roentgenograms

were negative. This was decided upon in the face of apparently negative findings, but with the previous history of an acute involvement of that ear, with loss of hearing, we thought that we were dealing with a comparatively rare unresolved mastoiditis with a healed middle ear. Upon opening the cortex, normal bone structure was encountered and only when the area of the aditus was exposed did we find about three drops of frank pus, which when cultured yielded the same organism that had been previously found in the blood stream and the opposite mastoid. The sinus and dural plate were removed and the underlying structures found healthy and were consequently not disturbed.

With a temperature drop lasting 24 hours postoperatively, we felt certain that the obscure focus had been discovered, but much to our chagrin the same picture reappeared soon after.

Tenderness persisted in the region of the right iliac fossa. The kidneys and ureters were ruled out as a possible focus of disease.

The surgeons were loath to explore the disputed region, but after two weeks of further observation explored the area of the head of the right femur and found an abscess of tremendous size. The upper third and head of the femur were literally bathed in pus, which was also of streptococcus hemolyticus origin.

In concluding, may I read the opinions of several consultants who saw the boy shortly before his final operation. In doing this I merely wish to bring out the difficulties that an otologist must combat at some time or other in his career.

1. June 3: Hypersensitiveness over several areas. Phlebitis of right leg. Slight if existant.

2. June 12: Patient very sensitive to palpation over entire body. Appears to be more sensitive over right iliac fossa and right hip joint. Patient complains of pain in hip on internal rotation of femur. Recommendations: *1.* Extension of right lower extremity; *2.* X-ray of right hip (this was essentially negative); *3.* Ice-cap on hip; *4.* Rectal examination for mass in right iliac fossa.

3. June 14: Rectal examination (report was negative).

4. June 15: Extreme tenderness over right kidney, anterior and posterior. The whole course of the ureter seems tender. History of burning urine (may be febrile). Chest negative except for dullness posteriorly in right base. No cardiac murmurs, but heart is enlarged to left—reacting to febrile course. Would watch for a perinephritic abscess.

5. June 16: Practically same condition as when seen on June 3. Blood stream infection can account for most of his symptoms.

6. June 16: No definite evidence of a perirenal abscess. However, X-ray of genitourinary tract for same will rule this out. (This was negative.) Believe that blood stream infection rather definitely accounts for patient's symptoms.

7. June 24: Examination of right hip (negative). No muscle spasm. Right ankle clonus present. History of projectile vomiting. Recommend examination of eyegrounds—cerebral abscess?

In conclusion, I am glad to say that after these months of vague symptoms and doubt, the patient made an uneventful recovery and is now receiving baking and massage to stimulate the return of function of his leg.

30 East 40th Street.

SYMPOSIUM ON SINUS THROMBOSIS.

II.—CASE OF RIGHT ACUTE MASTOIDITIS WITH LATERAL SINUS THROMBOSIS; SEPTICEMIA (BETAHEMOLYTIC STREPTOCOCCUS); OPERATION AND RECOVERY.*

DR. GERVAIS WARD MCAULIFFE, New York.

SUMMARY.

School boy, age 7 years, referred to New York Hospital because of persistent fever, headaches and peculiar spells of unconsciousness. Patient had enjoyed previous good health until about seven weeks before admission, when he suffered an acute attack of grippe, as did other members of the family. Three and one-half weeks before admission the child developed a very severe sore throat, generalized rash, glandular enlargement, light fever and other symptoms which now appear typical of scarlet fever. Two and one-half weeks before admission his right ear became tender, as well as the tip of the right mastoid. The drum head was incised and the tenderness disappeared. The chill then became afebrile and seemed to be getting well, when four days before admission, he had a severe chill and complained of pain over his eyes and gave general evidence of acute frontal sinusitis. He then began to run a septic temperature reaching as high as 105°. On the night before admission he became semicomatose, during which time he did not respond to questions. This attack lasted one-half hour and he returned to full consciousness and has had no similar seizure since then.

On admission (March 15, 1933) the child had slight fever, rapid pulse and appeared acutely ill. He was pallid and undernourished. The cheeks were flushed. The mouth was held open and he had what you might call a sinus facies. There was some tenderness along the supraorbital ridge. The pupils

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were equal. There was a tendency to jerky extraocular movements. The right fundus was negative. The left nerve head was definitely edematous on the nasal side with an increase in the vascular markings, some shimmering of the retina and photophobia. There was a clot of blood in the left E. A. C. The right drum head was perforated and drained a small amount of purulent material, though there was no tenderness over the right mastoid. There were numerous large tender glands beneath it. Throat was injected. Considerable postnasal discharge was present. Heart and lungs were clear. The spleen was palpable. There appeared to be a slight right facial weakness and the patient repeatedly pastpointed on the left side.

It was obvious that the patient had otitis media, probably postscarlatinal, and it was felt that in this case we should definitely rule out acute mastoiditis with lateral sinus thrombosis and also brain abscess. The patient was kept under close observation and began to run a septic fever which at first was not very marked. A spinal puncture was done on the day after admission and a normal pressure and cell count found, but no difference in the pressure on occlusion of either jugular vein. The urine was negative; the white blood count on admission 10,400 and two days later 22,400, with a moderate secondary anemia. During the first few days of admission the patient had a definite right facial weakness with nystagmus and a tendency to pastpoint. The first blood culture taken was negative. Another blood culture taken on March 24, was positive for betahemolytic streptococcus. On March 25, a spinal puncture done showed a positive Queckenstedt on the right side, with the fluid containing 25 cells, mostly lymphocytes. The culture was negative. Blood was then taken from both jugular veins and this on culture showed betahemolytic streptococcus. It was then decided to transfuse the boy and small transfusions of 150 cc. of citrated blood were given every few days. Six such transfusions were given. The right ear drum was reincised early in the hospital course and for a few days the right ear drained profusely. X-rays of the mastoids were taken and the right was found to be slightly cloudy.

Finally, on April 1, the child was taken to the operating room and under a general anesthetic the right mastoid was

opened. Some of the mastoid cells were definitely diseased and cultures from them later showed betahemolytic streptococcus. The lateral sinus was then dissected but no pulsations were felt and the sinus was capped off and completely exposed. Incision of the lateral sinus was followed by extrusion of a partly organized thrombus. The thrombus was carefully removed. The wound was loosely packed and left open. Following this operation ligation of the right jugular vein was done.

Following this operation the patient's temperature fell and in a few days came down almost to normal and he showed evidence of great improvement. It was necessary to dress the patient under gas twice with final removal of the packs which had been inserted, and a secondary closure of the wound was made. The wound healed rapidly. Blood culture taken immediately after operation showed only a few colonies, and two blood cultures taken thereafter were negative. The patient then went on to a remarkable recovery and was, 18 days after operation, discharged, apparently fully recovered.

110 West 55th Street.

SYMPOSIUM ON SINUS THROMBOSIS.

III.—PARAPHARYNGITIS FOLLOWING INFECTION OF THE LOWER JAW AFTER TOOTH EXTRACTION.*

DR. SAUL KNOPF, New York.

Notwithstanding the fact that this man's blood picture did not demonstrate any definite involvement to establish a classical diagnosis of internal jugular thrombosis, I feel with reasonable certainty that because of the circumstantial evidence surrounding the vein, a phlebitis did exist and further that the above pathological complication might have superimposed if complete and thorough drainage had not been ascertained by way of the most logical surgical exits emanating from the parapharyngeal, retropharyngeal and buccal areas, or as appropriately called, the pharyngomaxillary region. My opinion again is that the secret of successful recoveries from severe infections of the above locations is by far and large due to a procedure of securing adequate surgical drainage of the lymphatic channels by means of a detour along the carotid sheath, or as so well alluded to by Dr. Mosher as the "Lincoln Highway," so as to protect against involvement of the internal jugular and prevent serious systemic complications.

This patient, after three days of suffering from a persistent toothache, had a first right lower molar extracted which was performed under gas anesthesia because of his inability to completely and properly open his jaws. Although he had a slight swelling of his right lower jaw in the immediate vicinity of this tooth prior to the extraction, he noticed that a few hours later it increased in intensity and extended further down toward and under the angle of the jaw, with more definite pain in the wound. Later this swelling not only increased in amount, but also in extent, pursuing both an upward and downward course, especially prominent below the angle of the right lower jaw. Besides this fact he began to

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complain of inability to swallow food. Because of this alarming increasing difficulty associated with a marked general depression and high temperature, although there was no difficulty in breathing or speech, he was sent to the Nose and Throat Division of Dr. Harmon Smith at the Manhattan Eye, Ear, Nose and Throat Hospital, under my care.

He appeared very ill. His features were drawn and sallow. His right face and neck, particularly under the angle of the lower jaw, was swollen and tender, more exquisitely so, however, deeply along the anterior border of the sternomastoid along the course of the internal jugular down to the chest. The tenderness in the lower neck was confined to this location. He had great difficulty in opening his jaws, but notwithstanding this the tooth socket could be seen covered by a well organized exudate with a moderately congested tender swelling surrounding it and extending as a marked noninflammatory thick edema upward toward the ramus, right anterior pillar, soft palate and uvula, but with much less intensity beyond the median raphe. The right tonsil seemed to be pushed forward by a marked swelling in the right parapharyngeal region, which was also more like a thick edema rather than an actual inflammatory or suppurative lesion. He had very great difficulty in swallowing even fluids, a productive cough, but no distress in breathing or alteration in speech. His temperature upon admission was 103°, the next morning 102° and in the afternoon, just before operation, 104.3°, with the pulse 120. The blood picture revealed a normal blood chemistry, Type 4, negative Wassermann; R. B. C., 4,000,000; W. B. C., 15,800; polys., 92; S. L., 6; L. L., 2; E., none. On the day of operation, W. B. C. was 17,500, polys., 78, and X-rays of his jaw revealed some absorption around the right inferior molar, but no evidences of necrosis of the bone. The chest X-ray revealed small circumscribed areas of infiltration in the right lobe at the apex, suspicious of tuberculosis, but no tubercle bacilli were found in frequent sputums.

The afternoon following his admission he was brought to the operating room and prepared for local surgery. A barrage anesthesia with 1 per cent novocaine and adrenalin was administered, surrounding and including the lower half of the right face, mastoid and entire right neck with deeper injections wherever necessary, producing a complete anes-

thesia. An incision was made parallel to and just below the lower right jaw and perpendicular to this downward about three inches. It was interesting to note that on going through the skin and subcutaneous tissues the same thick edema was encountered (not due to the infiltration anesthesia, which was a barrage, but obviously due to a lymphatic involvement). Further dissection was performed down to the carotid sheath and an iodoform packing placed in the lower neck wound. Lying upon the sheath, on the vein and bifurcation of the carotid, beneath the styloid process, was an edematous lymphatic gland which was removed and examined, and which later revealed lymphoid hyperplasia, with distinct germ centers and marked epithelial cell hyperplasia with active mitosis. From this location I proceeded to strip the tissues along the sheath carefully to the base of the skull into the parapharyngeal region, along the tonsil and toward the floor of the mouth surrounding the mandible, but without finding any evidence of pus, only thick edema. The internal jugular, however, appeared normal, so that no further surgery was performed. However, the packing was replaced in the lower neck wound and drains inserted into each of the three pathways above mentioned. The wound was left open except the outer ends of the horizontal portion, and a wet dressing applied. The organism was streptococcus hemolyticus. Following this a deep incision was made in the right peritonsillar region and a curved artery forceps dipped behind the tonsil downward and spread carefully. However, even here no pus was discovered. The organism was a hemolytic streptococcus.

The patient was removed to the ward and rectal glucose 20 per cent was instilled for four hours and a donor was procured that evening. 700 cc. of blood was transfused, and six hours later another 600 cc. were given. Hot throat irrigations were ordered to be frequently repeated, and the tooth socket was irrigated a number of times a day. The outside dressing was also changed and the drains found soaked with a serosanguineous discharge, becoming mixed with a small amount of pus the next day. These drains, however, were not disturbed until the third day, when they were gradually removed and by the eighth day completely removed, and only a short drain reinserted for a number of days. The swelling on the side of his face and neck also gradually receded and by the end of the week had completely disappeared. His

swallowing and trismus gradually improved and by the end of the week was also normal. On the fifth day after the operation another X-ray of the jaw revealed an area of absorption extending from the root cavity of the first right inferior molar to the second inferior molar, which appeared to be an extension of a necrotic process. On the twelfth day his temperature was normal and the second molar tooth originally not involved was sufficiently loose to be easily extracted with an artery clamp. Almost immediately rapid improvement occurred. He was discharged on the twelfth day, feeling quite well and having gained a few pounds. Dressings were discarded a week later. Dental consultation and treatment was continued because of a slight tenderness at the ramus.

Two weeks later he returned because of a swelling which was rapidly increasing in the neck since two days previous, and because his jaw again became locked and pain in the region of the tooth socket recurred. He was admitted and an X-ray revealed an absorption of bone around the right first and second inferior tooth sockets and an area of sequestration about the size of a thumb nail in front of the ramus. He was taken to the operating room and under local anesthesia an incision was made at the site of the old operation and a very large quantity of pus evacuated. It was interesting to note that the pathways originally packed could be felt definitely by palpation with a finger. This abscess was cleansed by suction, lightly packed, and a wet dressing applied. Then with a large curette this small bony sequestrum in the jaw was removed. Within a few days the external wound and the wound in the jaw were completely healed and the trismus also completely relieved. No recurrences have presented themselves since.

17 East 38th Street.

SYMPOSIUM ON SINUS THROMBOSIS.

IV.—CASES OF JUGULAR THROMBOPHLEBITIS FOLLOWING INFECTIONS OF THE FACE AND THROAT.*

DR. JAMES W. BABCOCK, New York.

The following three cases of jugular thrombophlebitis following other than ear infection, occurring at Presbyterian Hospital, seemed of interest to report in connection with the papers of the evening.

Three cases, the infection arising from a different source in each, are reported.

Case 1: Miss B. B., age 27 years, a pharmacist, was admitted on April 14, 1930, with acute tonsillitis, left posterior deep cervical lymphadenitis and cellulitis of the left side of the neck. During the next few days this increased with involvement of the left carotid group of lymph nodes. Incision and drainage was performed on April 18. Relief was not complete, blood culture showed hemolytic streptococci and tenderness and infiltration was present along the course of the jugular vein to the clavicle. An operation was performed by Dr. Beverly Smith under rectal ether anesthesia on April 22, 1930. The left internal jugular vein was found collapsed but bled from both ends after removal of a thin thrombus two inches long and was doubly ligated and the portion between ligatures removed. Convalescence was stormy in that abscesses formed on right ankle and left arm at sites of puncture of veins, requiring incision and drainage, and a transient pericarditis developed. Transfusion was given and temperature fell to normal on May 26, 1930. Complete recovery ensued.

Case 2: Mrs. D. L. F., age 32 years, a housewife, was admitted on Oct. 22, 1931. A pimple had appeared on the right side of her chin nine days before admission and she had

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squeezed it. The inflammation extended and on admission there was swelling and one necrotic area on her chin, her lip and neck in the midline and right side were swollen, red and brawny two-thirds of the way to the clavicle, and the right eye was partially closed. There was no chemosis nor exophthalmos. Temperature was 103° F. Blood culture showed a slightly hemolytic staphylococcus aureus. Two days later there was tenderness over the lower third of the right sternocleidomastoid and some difficulty in opening the mouth attributed to involvement of the pterygoid plexus. Staphylococcus antitoxin was given intravenously. On Oct. 25, 1931, temperature was 105° F., and an operation was performed by Dr. John Hanford under rectal ether anesthesia. He ligated the right external and internal jugular veins through a horizontal incision just above the clavicle below the area of infiltration. A section from the internal jugular vein showed infiltration by polymorphonuclear leukocytes. The frontal vein was also explored. Bacteriophage was used locally and intravenously, and transfusion given. She was greatly improved after a stormy week and temperature was normal in two weeks, with full recovery.

Case 3: Mr. H. H., age 43 years, elevator operator, was admitted on March 17, 1932, with bronchopneumonia and suppurative pleurisy showing hemolytic staphylococcus aureus. Blood culture on March 25, showed hemolytic staphylococcus aureus. On March 28, a thoracostomy was performed under avertin anesthesia. On May 8, a tender red swelling was noted behind and above the right auricle with only a slight injection of right Shrapnell's membrane. On May 9, an operation was perfromed by Dr. George Brighton under avertin anesthesia. A small subperiosteal abscess was found at the site of the swelling, with a cortical perforation, leading to a perisinus abscess in the sinodural angle. The mastoid was sclerotic and the few cells present were not involved except right by the abscess. The sinus had no occluding thrombus, but it was deemed wiser to ligate the internal jugular vein. The patient did well and blood culture was sterile until June 5, when he complained of parosmia and nausea, was irritable and had a convulsion and right-sided weakness. He improved and no definite diagnosis of brain abscess was made. He was readmitted on Aug. 26, 1932, after frontal headaches of two weeks' duration and stupor on day of admission with choked

discs but no fever. A slight left facial weakness was present. On Aug. 29, an operation was performed by Dr. George Browne, who found and drained a right temporal lobe abscess. Meningitis followed, with death on Sept. 1, 1932. Hemolytic staphylococcus aureus was found in all the lesions.

These cases all arose from a different point of entry, but developed bacteremia and in two of them remote metastatic abscesses. In the last case the abscess in the brain proved fatal after seeming recovery.

20 East 53rd Street.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

At the Fortieth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., held at Charleston, S. C., April 3-5, 1934, Dr. Perry G. Goldsmith, Toronto, Canada, was elected President, and Dr. Robert L. Loughran, Bridgewater, Conn., Secretary, for the ensuing year. Date and location of next annual meeting will be determined later.

TREATMENT OF LATERAL SINUS PHLEBITIS, THROMBOSIS AND OTITIC SEPTICEMIA WITH NON-SPECIFIC IMMUNO TRANSFUSIONS.*†

DR. MATTHEW S. ERSNER and DR. DAVID MYERS,
Philadelphia.

The use of blood transfusion in the treatment of general sepsis of otitic origin has been generally accepted. Transfusion of whole blood, chemotherapy and sera has been used with varying degrees of success. In the treatment of sinus thrombosis and sinus phlebitis, surgery, combined with blood transfusion and other agents, has given favorable results.

In the treatment of septicemia, malignant endocarditis and other conditions, specific immuno transfusions have yielded encouraging results, as reported by Greenslade,[1] Brown,[2] Probstein and Seelig,[3] Kurtz and White,[4] and Brody and Crocker,[5] the latter who reported a case of hemolytic septicemia, treated with specific immuno transfusions plus chemotherapy with recovery.

Non-specific sera and vaccine have also been employed with favorable results in otitic septicemia.[6]

Stephenson[7] used non-specific immuno transfusion in a case of hemolytic streptococcus septicemia with recovery. This apparently is a contribution of great worth and adds a valuable agent to our therapeutic armamentorium, principally because of its availability without resorting to the aid of a bacteriologist or without waiting for results of blood cultures, the making of vaccines and the specific immunization of donors.

Sir Almroth Wright[8] states: "We should discard the confident, dogmatic belief that immunization must be specific."

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*Read before the October, 1933, meeting of the Otology Staff of Temple University.

†From the Department of Otology, Temple University School of Medicine.

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Following the report of Stephenson, we had the opportunity of treating a case of primary jugular bulb sinus phlebitis by this method. The convalescence was greatly hastened and the patient made an uneventful recovery. This leads us to report the following case because we believe that the method is worthy of use in the treatment of this dangerous complication:

W. F., age nine years, male. Patient was admitted to the Otologic Service at Temple University Hospital, March 10, 1933. Temperature on admission was 104.2° F.

Eight days previous to admission, the patient developed a cold and was in bed for four days. On the second day after the onset the patient experienced pain in the right ear, followed four hours later by a spontaneous perforation of the ear drum, with a purulent discharge. On the sixth day, the temperature rose to 103° F., and the following day to 105° F. On the eighth day, the patient was referred to the hospital for observation. At this time the chief symptom was headache.

The previous medical history was negative, except for an otitis media during early infancy, and tonsillectomy and adenoidectomy at the age of seven.

Physical examination revealed a bilateral central perforation with no discharge from either ear. The right ear drum was slightly thickened. Both mastoids were clinically free.

The pupils reacted equally to light and accommodation. Extra ocular muscle movements were good and equal in all directions.

Crowe-Beck sign was negative. Griesinger's sign was absent. No neurological signs were present. The eye grounds did not reveal any evidence of papilloedema, although a slight engorgement of the retinal veins was noted in the right eye.

X-ray examination revealed a slight increased density of cells on the right side without any evidence of bone destruction. It also showed the left lateral sinus and left jugular foramen to be much larger than the right.

A lumbar puncture was performed and the spinal fluid was negative; it was impossible at this time to perform the Tobey-Ayer test because the patient was extremely uncooperative.

In view of the above findings, the history of a suppurative otitis media, without definite evidence of mastoiditis, but with a septic temperature, the possibility of a primary jugular bulb thrombosis was considered (as described by McKernon[9]). However, it was decided to keep the child under observation temporarily.

On the evening of admission, the temperature rose to 106° F., with chills and sweats, and it was decided to explore the right mastoid and lateral sinus. A blood culture was made at this time and the findings were negative. The W.B.C. was 8,900. The Schilling index disclosed a definite shift to the left (see chart). The blood sedimentation test revealed a drop of 27 minims in 60 seconds (Cutler method).

On March 12, 1933, two days after admission and ten days after the onset of illness, the right mastoid was exposed and occasional isolated areas of pus and necrosis were found. The sinus plate seemed intact. The lateral sinus was then exposed from above the sigmoid to the jugular bulb. In the region of the bulb beneath the sinus plate, a small amount of pus was found. A culture made from the region of the bulb by venopuncture was found positive for streptococcus hemolyticus. Incision of the lateral sinus gave free bleeding from both ends of the sinus and no thrombus was found. The sinus was occluded by packing and the mastoid cavity was packed with iodoform gauze and the wound was then closed.

Immediately following the operation, a transfusion of 200 cc. of whole blood and 20 cc. of Pregl's iodine was given intravenously; the temperature then rose to 104° F. Subsequent treatment consisted of daily intravenous injections of 10 cc. of one-half per cent neutral acriflavine solution and 10 cc. of Pregl's iodine.

On March 14, 1933, two days after operation, a positive culture from the jugular bulb, as well as from the median basillic vein, was obtained. The temperature remained septic.

On March 16, 1933, four days after operation, non-specific immuno transfusion therapy was begun. The donor was prepared as suggested by Stephenson; a suitable typed and cross-agglutinated donor was put to bed and given intravenously one-tenth cc. of stock typhoid vaccine, containing fifty million killed organisms. This was followed by a sharp rise in temperature with chills and sweats. Seven hours later, the patient

was given 100 cc. of whole blood from the prepared donor by the direct syringe method and 100 cc. of blood was citrated and refrigerated and given to the patient after a lapse of sixteen hours.

There was no reaction noted following the non-specific immuno transfusion. The temperature reached a new level, 99.4° F., and the Schilling index showed a marked improvement with definite increase of segmented forms and lymphocytes.

On March 18, 1933, the sixth postoperative day, the temperature remained at 99°, with a marked improvement in the patient's condition. The wound was redressed and the packing obliterating the lateral sinus was loosened. This was followed by considerable bleeding. The wound was repacked and, as anticipated, the temperature rose again to 102.2° F. On the following day a further rise to 103.3° was noted.

On March 20, 1933, the eighth postoperative day, a non-specific immuno transfusion of 100 cc. from a new donor was given; a slight reaction, hardly worthy of mention, was noted. The temperature remained normal until March 22, when it rose to 101.2° F. Then gradually it became normal until March 24, the twelfth postoperative day, and continued normal until the patient's discharge from the hospital, forty days after admission. Intravenous medication and transfusion therapy was discontinued on the fifteenth postoperative day. The patient was placed on a high caloric diet and tonics, and made an uneventful recovery.

SUMMARY AND CONCLUSIONS.

1. This case presented considerable diagnostic difficulty. The fact that the trouble was on the right side primarily, with spontaneous perforation, then a cessation of the discharge with septicemia without a definite mastoiditis made the diagnosis of primary jugular bulb thrombosis most probable. A. A. Love states, "Careful evaluation of the atypical ear drum is necessary." We believe the diagnosis of primary jugular bulb and sinus phlebitis was warranted in view of the operative findings with the location of pus in the region of the jugular bulb.

2. The X-ray study was interesting. There was a slight increased density on the right side without evidence of bone

destruction. More important, however, was the fact that there was a large lateral sinus and jugular vein on the opposite side. As described by us in a previous communication, now in publication,[11] this insured an adequate drainage through the medium of the unaffected side and enabled us to prognosticate a more favorable outcome. In support of this is the fact that daily pre and postoperative eye ground studies showed no sign of choked disk. Even after surgical obliteration of the lateral sinus, daily eye ground studies failed to reveal any evidence of impaired venous circulation. This again illustrates the importance of an adequate contralateral circulation and the value of knowing this preoperatively.

3. Non-specific immuno transfusions were instituted four days after operation. The temperature was septic in character, with no abatement of the severe symptoms despite the operative intervention and whole blood transfusion and chemotherapy. The non-specific immuno transfusion, however, was followed by an immediate drop in the temperature and a favorable change in the Schilling index, with a beginning shift to the right. The immuno transfusion was repeated on the fifth and eighth postoperative days. The patient's physical condition and the Schilling index, being favorably influenced each time with a definite evidence of the shift to the right. At no time was there any severe reaction to the transfusion. The ease of preparing a donor and the definite aid that this method offers makes it worthy of trial.

SUMMARY OF BLOOD CHANGES—SCHILLING INDEX.

Date	Total W.B.C.	B	E	My	J	Stab.	Seg.	L	M
3/10	8,900	1	0	0	2	37	45	12	3
3/13	7,650	0	0	0	2	50	37	9	2
3/14	7,500	0	0	0	3	44	32	18	3
3/15	9,050	0	0	0	1	41	42	12	4
3/16	7,900	0	0	0	2	32	36	25	5
3/17	12,600	0	0	0	1	23	41	34	1
3/18	8,200	0	0	0	0	34	26	34	4
3/20	14,150	0	0	0	2	27	42	26	3
3/21	10,900	0	0	0	2	31	44	18	5
4/12	10,800	0	2	0	0	9	42	44	3
4/20	8,900	1	3	0	0	9	44	34	9

W.B.C.—white blood count.
B —basophiles.
E —eosinophiles.
My —N. myelocytes.
J —N. juveniles.
Stab.—
Seg. —segmented.
L —lymphocytes.
M —monocytes.

Fig. 1.

4. Blood work. It was noted that the total leukocyte count ranged from eight to fourteen thousand. The Schilling index, however, is most valuable, as it gives one a daily index of the patient's resistance to the infection and therefore should be utilized. The daily blood sedimentation rate did not show any definite drop and it was useless in the prognostication of the severity of illness because the variation curve did not coincide with the temperature, Schilling index or the general improvement of the patient.

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GROSS PATHOLOGICAL CHANGES IN MASTOIDS ON VARIOUS DAYS FOLLOWING THE ONSET OF ACUTE SUPPURATIVE OTITIS MEDIA.

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In an attempt to visualize what takes place within the mastoid on successive days following the onset of acute suppurative otitis media, we have attempted to analyze our findings in 25 cases which came to operation. The ages of these patients varied from five to 58 years. Only those of five years or over were selected, in order that we might deal with the adult type of mastoid.

The time of operation from the onset of the acute otitis media varied from the third to the fifty-fifth day and may be tabulated as follows:

Third day	2	Twelfth day	2
Fourth day	1	Eighteenth day	1
Sixth day	3	Nineteenth day	2
Seventh day	2	Twenty-first day	1
Eighth day	4	Twenty-second day	2
Ninth day	1	Twenty-ninth day	1
Tenth day	1	Thirtieth day	1
Fifty-fifth day	1		

In this tabulation the cases fall naturally into two groups: The acute cases from the third to twelfth day, and the subacute from the eighteenth to the fifty-fifth. It is interesting to note that there were no cases demanding immediate operation between the twelfth and eighteenth days.

In the group of 16 acute cases, all began with well defined symptoms of mastoid involvement and had shown but little improvement or had developed more unfavorable symptoms at the time of operation.

Four of these, or 25 per cent, showed discoloration and softening of the periosteum and cortex. The earliest that this

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was noted was on the eighth day in a man, age 58 years, with a virulent streptococcic infection. On the ninth day, a boy, age 12 years, had a perforation of the cortex with subperiosteal abscess. On the twelfth day, in a girl, age 6 years, there was found to be a marked softening and discoloration of the periosteum and cortex of both mastoids.

Four others of this group had sclerotic mastoids. It is noteworthy that of three complications occurring in this group, all were this type of mastoid. These complications were as follows: Perisinus abscess and lateral sinus thrombosis, two; localized meningitis, one.

The two cases of sinus thrombosis were operated on for acute mastoiditis on the sixth day following the otitis media, at which time there was no apparent disease of the bony covering of the sinus. Both were operated upon for the complication on the thirteenth day, when there was decided softening of the bony wall.

The patient with localized meningitis was admitted to the hospital on the seventh day following her acute otitis. This day marked the onset of both an acute mastoiditis and symptoms of meningeal irritation. Following operation on the day of admission, she remained in a semicomatose condition with intervals of acute delirium for three days, then her mental condition gradually cleared up and she made an uneventful recovery.

In this group, the findings within the mastoid were such as would be expected from the severity of the symptoms. Hyperemia, pus under pressure, polypoid formation and decided softening of the septa were encountered in children of $6\frac{1}{2}$ and 14 years, respectively, as early as the third day after the onset of the acute otitis. Actual breaking down with cavity formation was present in a girl of 5 years as early as the fifth day. Up to the twelfth day, however, no actual uncovering of the lateral sinus or dura by necrosis of the bone was encountered.

A rather constant and most interesting finding from the sixth day on, was the presence of a large firm mass of polypoid tissue almost completely blocking the mastoid antrum. This corresponded clinically in each case with the decrease or cessation of the discharge from the middle ear in spite of a free

opening in the drum, together with an exacerbation of the fever, pain and mastoid tenderness. It would seem that this was nature's attempt to limit infection to the middle ear, but occurred too late to be effective and by preventing adequate drainage from the mastoid, accelerated the progress of the pathological changes.

The second or subacute group comprises those cases operated upon from the eighteenth to the fifty-fifth day. These patients had shown steady improvement and were considered to have a good chance for recovery until sudden unfavorable symptoms appeared which made operation imperative. There were nine such cases in this group.

Discoloration and softening of the cortex was noted but once, and that on the fifty-fifth day.

Two cases had a marked sclerosis of the mastoid. One developed a well defined Gradenigo's syndrome on the twenty-ninth day, which, however, cleared up promptly following operation. The other did not develop any complication.

Free pus, polypoid formation and softening of the septa were present in all cases, although necrosis of the bone with exposure of the lateral sinus was present in but two, one on the eighteenth, the other on the fifty-fifth day.

The only complication in a nonsclerotic mastoid in either group occurred in a woman of 35 years, who was in her ninth month of pregnancy. She had been under observation for two weeks following an acute otitis media and had been allowed to return to her home in the country under the care of her physician. On the fifty-third day, she developed symptoms of acute labyrinthitis with severe vertigo, staggering, falling, nausea and vomiting, and nystagmus.

She was admitted to the hospital the same day, delivered of a normal infant the next, and operated upon for her mastoiditis on the third day. After recovery from the anesthetic, her labyrinthine symptoms were entirely relieved, and in spite of a rather extensive necrotic exposure of the lateral sinus and dura, she made an uneventful recovery.

While it is fully realized that definite conclusions can not be drawn from such a small number of cases, the findings in

this series seems to emphasize several points of practical importance:

1. That in acute mastoiditis, marked pathological changes may be present in the mastoid as early as the third day following the onset of an acute suppurative otitis media.

2. That the rapidity of bone destruction depends to a great extent upon the age of the patient, being more rapid in the young, and also upon the severity of the clinical symptoms.

3. That in this series, four out of five complications occurred in patients with sclerotic mastoids. This type of mastoid can be readily detected by X-ray photography and would seem to be an indication for early operation.

4. That some cases of acute labyrinthitis, Gradenigo's syndrome and meningeal irritation may recover without other interference than the simple mastoid operation.

5. That a sudden decrease or cessation of discharge from the middle ear, in the presence of a free opening in the drum, and with an exacerbation of clinical symptoms, nearly always means complete blocking of the mastoid antrum, which can only be relieved by operation.

6. That patients who continue to show signs of mastoid involvement as late as the eighteenth day following the onset of an acute suppurative otitis media, even without marked evidence of bone destruction, as shown by the X-ray, have sufficient pathology within the mastoid to make satisfactory recovery without operation extremely doubtful.

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OTITIC GENERALIZED INFECTION; ORIGIN; MANAGEMENT; OUTCOME.

DR. ARTHUR KOVACS, Milwaukee.

The source of systemic infection of otitic origin in most cases can be found in the thrombotic process of dural sinuses that are first exposed to the infection that originates in the middle ear region. Through the transmission of infected thrombus particles or through repeated bacterial aggression, pathogenic organisms invade the circulation.

In the formation of the thrombus the inflammations of the vessel wall, especially that of the intima, play important roles. The dura which forms the vessel wall is only half thick and thus if the infectious material stays, for any length of time, in contact with the same, the penetration is promoted.

The septic process may be brought about by the infection of the fine vessel system in the septa of the pneumatic cells or of those that empty into the sinus, before sufficient time for formation of macroscopically noticeable thrombus had elapsed. These minute vessels necessarily get diseased when passing through a bone, in the process of purulent disintegration; the outcome will be phlebitis and periphlebitis.

Case of this type was observed and treated by me when a baby girl, 9 months old, exhibited signs of bacteremia, originating from an acutely diseased mastoid process. Germs have swarmed the cerebrospinal liquor and blood, setting up inflammation in the right ocular chamber, in the small digital joints, manifesting in erythema of the skin. On operation free pus in cells and antrum, but normal encasement of sinus was found. Rapid recovery on exenteration of cells followed. Uncovering of the vein has been omitted. This case corresponds with the osteophlebitis pyemia described by Körner.

According to Wittmack, the absorption of germs is facilitated from the wide, ramified, thin-walled vessel capillaries in the subepithelial mucous membrane and in the bone cavities, *i. e.*, in final instance through capillar thromboses. This origin must be taken as granted, in want of bone pathology, reaching to the sinus, in contrast to the contact infection when

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the mastoid process directly transmits the disease on the sinus parieta.

The genesis of otitic systemic infection may lie in the injuries of the vessel wall during operation, when after one-two weeks of interval, the normal progress of recovery is suddenly arrested by symptoms of a grave acute exacerbation. It is more common that an infection, latent before operation, becomes manifest through the operative trauma and the feverless patient, right after the surgery, shoots up a high temperature. The differentiation of this latter, so called latent sinus thrombosis from the aforementioned, postoperative, is exceedingly important from the practical standpoint, as the prognosis of the postoperative sinus thrombosis is very grave and it requires a very active therapy. From Brunner's series of six cases, only one has recovered. According to Bondy, the postoperative sinus thrombosis is characterized by the following signs: no indication of a previously existing sinus thrombosis, on first exposure normal appearance of sinus, pyogenic temperature after a given interval and thrombosis at the site exposed. In a case of latent sinus thrombosis the temperature elevation manifests itself right after the operation and the prognosis is much better.

The different origin of the otitic sepsis or pyemia makes evident that routine procedure in the management of the different cases would constitute a great mistake; the surgeon who would ligate the jugular vein in every case would commit the same failure as that one who is expecting a spontaneous recovery.

Körner and others have experienced that the sinus, accidentally put free during mastoid operation, became the source of generalized infection, by secundary thrombotic process of the exposed part. Leichsenring collected 17 similar cases from the literature. I had the opportunity to observe a fatal incidence of same origin, in the otologic department of an Austrian military hospital in 1918. The exposed sinus particle was the size of a pea, apparently perfectly normal. No puncture has been attempted, as no sign of inflammation was present. The progress was undisturbed for two weeks, when sudden profuse bleeding and marked chills appeared. Prompt intervention, with obliteration of the jugular vein, was of no avail.

The exposure of the vessel injures the superficial layers,

thus creating a tendency for thrombosis. Besides the exposure the presence of highly virulent germs and stagnation of secretion foster the development in this direction. Kriegsman undertook the task of analyzing 241 cases of antrotomies and 86 of radical mastoids. He arrives at the conclusion that uncovering of the sigmoid sinus is more apt to invite danger than generally accepted. He also finds that early operated cases are the most liable to undergo this complication, and for this reason advocates to refrain from the liberation of the sinus in uncomplicated mastoids. In chronic cases, those in the stage of acute exacerbation appear the most dangerous. There are a number of publications where the contact of the vessel wall with the plastic flap was made responsible; therefore, open treatment of the wound was advocated, while in antrotomies, the frequent change of tampon covering the sinus seems the best precautionary measure. Kriegsman argues that exposure of the vein, without well founded suspicion for disease, should be discarded.

While puncture of the sinus for diagnostic purposes or incision of the same, practiced by some otologists, is considered as harmless if proper precautions were employed, circulation of micro-organisms in the blood stream, however, alters the aspect altogether. Brieger has noticed that in case of general pyemia the sinus wall reacts on minor surgical procedures (incision, trial punction) with the formation of parietal thrombus, thus the clot formation often appears rather as a consequence than a cause of the systemic infection. Hayman made similar observations in his experiments when puncture or incision of the sinus, in existing bacteremia, has led to formation of infected blood coagulum. Operative trauma and exploration were responsible for the aggravated condition, after radical mastoidectomy, in a case of chronic purulent otitis, complicated with cholesteatoma formation.

The nine-year-old girl, Gladys G., seen on Jan. 10, 1929, had had otitis media on both sides since she suffered with scarlet fever five years previously. For the past three weeks, child had run mild temperature, lost appetite and became very fatigued. Examination revealed left, large central perforation of drum, pus from the middle ear and Eustachian tube. Right external meatus at fundus filled with polyps and fetid discharge. Mastoid process sensitive on pressure.

Hearing reduced in both ears, more so left. Temperature 99°. In face of this clinical picture, the culpability of the right middle ear was evident. The central perforation of the left drum excluded that cavity as a responsible agent for the complicating disease.

In following two weeks general condition, sensitiveness of mastoid process gradually grew worse, temperature elevation moderate, but always present at the time of treatment. No chills. Radical mastoid operation right, on Jan. 26, 1929. Cholesteatoma formation was found and free sinus on puncture of which blood withdrawn.

Following the operation, girl became gravely ill, with swinging temperature, indicating septic absorption. The case gradually resolved with lytic descendance of the pyrexia. In this instance phlebitis, probable parietal thrombus account for the sequelae. Pathologic permeability of the vessel walls yielded entrance to the germs.

Clinical observation and experimental research attest to feasibility of pyemia without thrombus formation when the putrefacting agents are swept into the circulation direct from the vessel wall.

Trying condition of a latent sinus thrombosis has developed in another instance where only the anamnesis, in the light of the sudden aggravation after the exenteration, sheds proper light on the pathology. The brief history is as follows: D. K., seen on May 23, 1929, had been ailing the last four weeks with right ear pain and discharge. The day before house physician found high temperature. Child apparently well at the time of examination. No pain on pressure of right mastoid process nor any discoloration or swelling of soft parts. Drum reddish, modestly bulging, scant mucopurulent discharge from tympanum. Temperature 100.5°. On the ground of the objective symptoms a harmless ear condition diagnosed. Following day paracentesis made, as drum was bulging; trace of mucus discharged. Patient presented again four days later, when mother relates the child at times shows peculiar behaviour, changed character, then severe attack of headaches distresses her, radiating down the neck and shoulder. After onset girl boon and playful. On examination middle ear healing, good hearing, whispering at two meter distance, trace of serous outflow. Fovea mastoidea sensitive. Temperature 100.5°.

On May 29, tenderness of mastoid more marked. Temperature 100°. Following day operation. Pulsating pus under pressure. Sinus in advanced position, of conspicuously large dimension, dominating the whole mastoid process, extending into tip, closely adapted to the antrum. Subdural cells show no pus. On elevating the sinus wall in order to inspect apex, moderate bleeding therefrom. Repeated bleeding from the sinus, manipulating around it in attempt to reach antrum. The same night patient restless, awake, vomited several times. May 31: Suffering, at times crying, irrational; complains of headache, unable to keep even liquid. Temperature up to 105°. No rigor or profuse sweating, tongue dry coated. Kernig, Babinsky, Oppenheim negative. Stiffness of the neck absent.

Subsequently it has developed a most exasperating condition. While the wound has shown a nice progress, rapidly granulating and sinus wall of healthy, pinkish color, daily pyemic attacks were at order, with furibund deliria. The stuporous condition alternated with epochs of agitation, jactation, efforts to jump from the bed and tear off the dressing.

Keeping in mind the aforesaid danger, on puncture of sinus, in pyemic condition, manipulation on latter was entirely omitted. It was deemed advisable to follow an expectative course for two reasons: the parietal thrombus, once the surrounding and initial focus infection is evacuated is capable of spontaneous recovery, therefore one should wait for the eventuality of such favorable change; the other reason, advocating such attitude is the fact that the management of these cases requires the transformation of the parietal thrombus into an obturating one; in other words, the recovery is attainable only through a temporary spreading of the infection, through an added risk. However, our patience, in administering merely internal treatment was about exhausted when the illness took a fortunate turn, a week later, with rapid recovery thereafter. This history shows an acute exacerbation of a systemic ear condition, right after surgery. The bleeding, during operation, was a sign of the lowered resistance of the sinus wall. The peculiar behavior before operation, related by the mother, was due to inception of the generalized disease.

In contrast to these cases, with fortunate outcomes, I hereby refer to two fatal cases: One, a chronic otitic sepsis, published in particular elsewhere, where the delayed interven-

tion, exhaustion of resistance, indication of lung metastasis at the time of surgery, carried out four weeks after the onset of the bacteremia, were responsible for the lethal end.

In another instance, a 19 year old, vigorous young man, operated in the third week of his otitis media, exhibited necrotic mastoid process with normal capsule of the sinus. As symptoms of bacteremia were present after the antrotomy, further interference was planned, but was prevented. By exclusion of other clinical possibilities, the operative finding, finally the rapid spread of infection, early metastasis in lung, in itself unusual in acute involvements, left no doubt in my mind, in spite of lack of pathological proof that this was a case of primary jugular bulb thrombosis. In these two cases our present day rational treatment was frustrated. In carrying out the latter, the ligature of the jugular, first recommended about 40 years ago by Zaufal, signifies a turning point in the management of otitic phlebothrombosis.

However, in spite of the long period which has elapsed and the ample opportunity to test its merit and efficacy, the opinion of the various authors is still divergent. While one group of otologists advocate the ligature, in every case of systemic infection, more than that, some of them previously to the mastoid operation, others advise this procedure only under certain circumstances. They argue that the ligature is not a safe method in preventing the expansion of the infection; they call attention to the venous connection of the sigmoid and bulbus (super and infer. petrosal sinus, post, condyloid emissary to the vertebral plexus); on the other hand, some danger is undeniably connected with the disturbance of the circulation. My former association with one of the protagonists of Zaufal's method and reading some of the case histories of the late Alexander, who belonged to the same liberal group, convinced me of the fact that undertaking the ligature under definite indication assures better result. These indications are as follows: Ligature of the jugular in case of disease of the vein itself; presence of primary or secondary obturating bulbus thrombosis, with grave general symptoms; when the central end of the thrombus is reachable, however, thorough removal impossible, due to bleeding from the bulbar end; parietal thrombus with manifestation of systemic infection and no tendency for spontaneous recovery.

This short series of five cases present an interesting variety of systemic infections of otogenous origin. The conclusions drawn from these and large material of prominent specialists prove that different origin (acute or chronic disease), different underlying pathology point to different prognosis and this should be kept in mind in combating the disease, which still shows a high mortality rate in the best hands of our profession.

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161 West Wisconsin Avenue.

A CONSIDERATION OF GENUINE OZENA.*

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In conversation with medical associates on various occasions, I have been astounded at the oft-repeated statement that ozena is comparatively rare. I cannot subscribe to such belief, for my own experience has shown it definitely otherwise and a review of the literature, especially that issuing from the pens of southern European investigators, totally refutes this optimistic point of view. In looking over records of my own cases, I find that the afflicted have most frequently been natives of or descendants of natives of southern and southeastern Europe. Literature from Spain shows the condition to be very common there, but I personally happen to have had but very few patients of that nationality. That atrophic rhinitis or ozena often fails to arouse interest in the medical profession may be ascribed to the fact that this disease entity is not dangerous to life, does not produce pain, is not disfiguring and in the great majority of cases, the physical status is only slightly, if at all, impaired. However, that ozena does give rise to such symptoms as tend to make it a very serious nuisance to the individual afflicted is attested to by the numerous publications on this subject, each author striving earnestly to bring to light new data on the etiology and therapy. I am purposely omitting any discussion of the signs, symptoms and diagnosis of ozena, since it would be absolutely superfluous before a group of rhinologists. In using the terms "atrophic rhinitis" and "ozena" interchangeably, I subscribe to the concensus of opinion of most rhinologists that they are one and the same disease entity, ozena being a more advanced stage of the condition.

With reference to the age and sex incidence of the disease, I have found a preponderance of females in the ratio of eight to one. Leroux-Roberts of Paris reports its presence in three females to every male, but taking into consideration the percentages of the majority of reports, I would state that the

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average is about five females to every male. While it has been reported that young children have been affected, the usual age incidence is in the second decade, with rare exception does the onset occur after age 45 years. In southern climates genuine ozena makes its appearance earlier as a rule. No case of genuine ozena at birth has ever been reported and while it is true that in several instances the symptoms of ozena were found present in infants, later evidence disclosed these to be on a luetic basis. Halle states that he has reports from Brazilian and Portugese rhinologists of ozena at age 2 years, syphilis, tuberculosis or other constitutional factors being ruled out. In my own experience, the youngest patient was age 5 years. The majority of cases seen occur at the age of puberty, but if one goes thoroughly into the history of these patients, one finds that there has been a nasal discharge for many years previously, so that it is very difficult to ascertain the exact age when symptoms first presented themselves. From a survey of the majority of published reports, there appears to be no question of the presence of a hereditary factor. Quite often it has been shown that the disease can be traced through several generations and in view of the pathologicoanatomic studies which I shall describe later, it seems fairly reasonable to assume that it is the predisposition that is inherited rather than the disease itself. My own records show many instances of several cases in one family as well as a fewer number in which I was able to trace it through three generations. On the other hand, Lermoyez, Bŏssowski and Viggo Schmidt have reported a few cases in which the disease was probably transmitted from one mate to another, but there may be a question of doubt as to the presence of lues in these cases. Perez reports an incident in which ozena was supposedly transmitted from a wet-nurse to an infant, but when the child came under the treatment of another physician years later, examination revealed that it was due to a syphilitic infection. In order to refute the possibility of transmission of genuine ozena from one individual to another, Costiniu and St. Popian carried out a series of experiments along this line in 1931. They first attempted to transmit the disease from man to animals, such as dogs, cats, etc., but without success. Then they chose 12 individuals free of any ear, nose or throat pathology. These included both sexes of various ages, ranging from 17 to 42 years, and all were kept under observation approximately 12 months following the

experiment. In the first three cases, the mucus membranes of both inferior turbinates were smeared with crusts from ozena patients. A local reaction resembling acute rhinitis with slight elevation of temperature, occurred in each case and all reaction subsided within four to six weeks. In the next four cases tampons soaked in an emulsion from the crusts of ozena patients were placed in the nasal cavities for 24 hours and in two of these patients the mucus membrane was purposely traumatized. The local and general reaction was more severe in the latter cases, but within a period ranging from ten days to three months, all reactions subsided. In the third series of two cases an emulsion of the ozena crusts was injected under the mucus membrane of the inferior turbinate. In the remaining three patients a similar procedure was carried out except that a bacterial emulsion was employed. The patients in series three and four reacted very violently to the infections, with high temperature, headaches, chills, nausea and vomiting, which lasted from three to five days. The mucus membrane was intensely congested and on pressure some pus came from the site of inoculation. The organisms recovered from this pus were the same as those injected, namely: Lowenberg, Abel, Hoffman, Perez and Mucosus Capsulatus. After several weeeks all signs and symptoms disappeared. Throughout the entire experiment there was no recurrence of symptoms in any case during the period of observation. From these results the authors conclude that ozena is in no way infectious and none of the organisms are specific for the disease. I feel that their findings are so conclusive that we may now definitely discard the theory of infection. In connection with the discussion of the theory of infection and contagion, I should like to enumerate some of the various causative factors which have been heretofore propounded. This subject was very thoroughly covered by Dr. Lee Hurd in his paper recently presented in Boston and I shall quote in brief some of the theories proposed to date. Probably the earliest was that of Grünwald and his school, who believed that ozena was the result of chronic paranasal sinus infection. In these cases a hypertrophic stage usually preceded the atrophy. Frankel claims that chronic simple inflammation of the Schneiderian membrane, with its subsequent degeneration, plays a great role in the establishment of ozena. The trophoneurotic theory of Zarnicko, which has for its basis a destruction of the nerve center of the nose, has

received rigid and almost conclusive support, but, as I shall later show, has not in the light of newer investigations, held its ground. That lack of vitamin could produce ozena was strongly urged by Glasschieb. However, since this is an exogenous factor, it stands in strong apposition to any inherited, developmental defect and so has very little basis. Furthermore, genuine ozena has rarely been seen in avitaminosis Halphen and Schulmann support the sympathetic endocrine theory which attributes the cause to a sympathetic disease of endocrine origin. The local character of the lesion and the limitation of the bone changes strongly refute this possibility. The bone theory of Zaufal which, in reality, is the mechanical theory, places the responsibility for ozena on the congenital, abnormally wide nasal cavities. No attention is paid to the mucosa, which factor should be taken into account when one realizes that there are many wide nasal cavities without ozena.

Enlarging on Zaufal's theory, Otto Fleischmann ably presents extensive data based on macro and micropathologico-anatomic studies of the bone and mucosa of genuine ozena. He concludes that it is an inherited lack of development of the bones and mucosa of the nose which places the stigma of a predisposition on these patients. These important studies are so clear and definite that I deem it essential to describe in some detail this work which has been carried out by such able investigators as Schuchart, Demme, Rethi, Seifer, Cordes, Siebenmann, Wright, Lautenschlager, Hofer, Sternberg and Saxon. Considering the macroscopical bony changes, we find that the bony framework of the nose is not fully developed, that is, of infantile type. Associated with this are changes in the cartilage, producing a typical ozena saddle nose, characterized by a deep-seated nasal root, a wide flat nasal dorsum and the apex tilted upwards. The nostrils are relatively wide and the soft parts thickened. The percentage of these changes in genuine ozena patients is approximately 40 to 80 per cent. At the same time there is arrested development of the nasal septum and hard palate which gives rise to a shortened septum and a short, broad, hard palate. Associated with some true ozena cases is a marked diminution of the sweat glands of the body. A majority of cases show not only infantile nasal structure, but also an infantile type of the skull as a whole. Zuckerkandl found a marked alteration in the development of the turbinates in that the bone at the onset of the atrophy is usually thin, brittle and perforated in several places. In

later stages a definite change in the contour of the turbinates is produced by a continual formation of dehiscences so that the turbinates become shorter and narrower until finally all that is left is a narrow ledge along the lateral wall of the nose. The cartilage of the nose is usually markedly soft. Siebenmann found the auricular cartilage also soft in these cases and believes that it is due to a general maldevelopment. As a rule the sinuses show a partial lack of development in about 30 per cent of cases and the mastoid air cells are poorly pneumatized in approximately 50 per cent of cases seen. Macroscopical abnormalities of a similar nature have not been found in other bones of the body. Microscopically, the bones in the region of the turbinates are only fairly well developed, their surfaces but partially smooth, the remainder being jagged due to numerous Howschip's Lacunae. In the latter, as well as in the marrow spaces, many osteoclasts are found, but osteoblasts are few. As a result, bony laminae are thin and the marrow spaces are enlarged and filled with cell-poor connective tissue with more or less abundance of plasma cells. Similar changes of lacunar arrosion were found in the bony septum by Krause; Lautenschlager confirmed this finding in the medial and facial walls of the maxilla. These changes as described are not of inflammatory nature, but are associated with lack of proper development with or without early senile changes. The macroscopic alteration of the mucosa of the nasal cavity usually presents a furrowed, shrunken appearance, is freely movable, pale, thin and solid. This is variable, since Lautenschlager has shown the mucosa, in some cases, to be loose and thick. The mucosa of the sinuses is thin and quite adherent, although edematous in the early stages.

The microscopic appearance of the mucosa is more characteristic than the macroscopic. The ciliated columnar epithelium in the nasal cavities is replaced by squamous epithelium which shows some hornification. In the subepithelial tissue there is an infiltration of round and plasma cells. These are more abundant in the earlier stages of the disease. Associated with metaplastic changes of the epithelium is the increase of fibrous tissue in the subepithelial layer which accounts for the shrinking. The vessels do not show any specific change, but in some cases endarteritis obliterans was found. The cavernous spaces are collapsed and the gland cells degenerated. The

mucosae of the sinuses show changes similar to that of the nasal cavities, but frequent capillary hemorrhages are very striking. On the whole, such mucosal changes of the nose and adnexa are not specific for genuine ozena, as they may occur in senile and secondary forms of that condition. However, these changes are most likely associated with a lack of proper development of the mucosa or a premature senile alteration. Results of these investigations of the mucosa do not substantiate a developmental defect so clearly as does the change in the cranial bones. The above-mentioned significance of the pathologicoanatomic findings in genuine ozena is truly not demonstrated in every instance, but offers a more conclusive basis than do other etiological explanations.

So far as body chemistry is concerned, the results do not speak for any specific relationship to ozena. Some authors have reported a decreased cholesterin content of the blood, but this finding has not received definite confirmation, at least not sufficient to warrant a dogmatic statement.

A study of the neuropathology in cases of genuine ozena has been productive of various interesting conclusions, the salient points of which I should like to mention in brief. There can be no doubt that a lesion of the nerve center of the nose is in some way associated with changes occurring in the nasal structures. That primary nerve changes result in ozena has been given support by a great many rhinological investigators, yet, on the other hand, a fewer number support the theory that nerve alterations associated with genuine ozena are of secondary nature. Worms has twice seen the occurrence of atrophy of the nose following a paralysis of the fifth nerve and Reverchon reports the presence of a unilateral ozena in a patient who, 18 months previously, had suffered injury to the region of the sphenopalatine ganglion. Helsmoortel, Rebattu and Proby, as well as Belinoff, have reported cases of unilateral ozena following shortly after injury to the sphenopalatine ganglion. Experiments have been carried out to determine the effect, if any, of extirpation of the sphenopalatine ganglion in man and animals, and results have not been at all uniform. In some, atrophy did occur, but a larger number showed no positive findings. One definite conclusion can be arrived at from the extensive resumé of Vogel's work on the sphenopalatine ganglion and that is: In genuine atrophic rhinitis the ganglion does invariably show definite

changes of a degenerative nature. As to whether these alterations are primary or secondary, it is impossible to say as yet. Since both primary and secondary degeneration have had some clinical and histopathological foundation, we must assume that another factor comes into play. Having in mind these results as well as those of the pathologicoanatomic studies of the cranial bones and mucosa, I wish to submit my version of the etiology of genuine atrophic rhinitis.

An individual born with the pathologicoanatomic changes of the cranium heretofore described is predisposed to the condition, but the presence of an exciting factor is necessary to its development and accompanying symptoms. By "exciting factor" I have reference to a sinusitis particularly, or a general infectious disease, such as scarlet fever, diphtheria, etc., with a consequent degeneration of the sphenopalatine ganglion, leading to neurotrophic disturbances in the nose and the formation of the disease entity, ozena. Since the structures of the nose have been shown to be involved in a defective development, we may reasonably assume that the nerve center of the nose, *viz.*, the sphenopalatine ganglion, is likewise affected and as a result possesses an inherent lack of resistance. Is it not probable that in the experiments of extirpating the sphenopalatine ganglion, ozena resulted only in those few individuals of a predisposed type, while the larger number not affected were not so predisposed. Furthermore, of the many thousands of young people afflicted with severe sinusitis, very few in your experience or mine develop genuine ozena as a sequela. A predisposition must be present.

As important as the investigations and studies of the etiology of atrophic rhinitis may be, from a practical standpoint what concerns us most is the alleviation and cure of this most annoying disease. In outlining the various methods of treatment employed in atrophic rhinitis, I would divide these into two major classifications, depending upon the stage of the disease when the patient is first seen. In my own practice, 95 per cent first present themselves when the symptoms are far advanced, *i.e.*, blockage of the nose, foul odor, expelling of large crusts, dry throat and often hoarseness. The remaining 5 per cent are seen before definite atrophic changes have occurred, and they comprise children up to the age of 11 years with complaints of thick, mucoid nasal discharge and nasal

obstruction. These symptoms persist rather constantly regardless of weather or season. Upon examination a somewhat typical ozena facies, as described earlier in this paper, is observed. The mucus membranes appear thickened and bathed with abundant mucoid secretion and in spite of an enlargement of the intranasal structures, there is a definite increase in the intranasal space. Detailed examination, including transillumination and skiographs, has failed in the great majority of instances to disclose the presence of sinus disease. Relative to these early cases I wish to emphasize that I am firmly convinced that if proper local nasal treatment is instituted, the progress of the disease can be checked. Concerning the therapy of the advanced cases you have undoubtedly many times heard and probably will hear again of the value of palliative treatment, yet it has been my experience and that of others that while it may give some relief, the alleviation of symptoms by such method is never permanent. Cleansing the nose with various douches and the use of suction give only temporary relief for crusting and odor, and should not be relied upon as giving hope of a cure.

During the past five years reports on the use of acetylcholine locally with or without the combined use of pilocarpine hypodermically, have appeared in the Belgian and German literature. Guns and Coene, in 1929, and Guns and Picard, in 1933, published their results on the use of acetylcholine and pilocarpine in genuine ozena cases. They based this form of therapy on their assumption that changes in the fibers of the vegetative nervous system produce trophic disturbances in the nose with consequent formation of the clinical picture of ozena. They claim that acetylcholine produces a vasodilatation and reactivity of the mucus glands, thus bringing about an alleviation in the symptoms by creation of an almost normal mucosa. In combination with acetylcholine and pilocarpine, they also employed tamponade with lactic acid solution with the following results: 75 per cent cured with acetylcholine alone; 15 per cent additional cures with the combined use of acetylcholine and pilocarpine; the remaining 10 per cent were cured by the Halle-Lautenschlager operation. This is the first time that I have had occasion to note a report of 100 per cent cure. However, in May, 1933, Kobrak reported his attempt to corroborate these results by using the same medicaments and although he obtained some improvement in

the symptoms, he does not believe that it was due to any specific action of the vasodilator. He attributes the alleviation of the symptoms, which were temporary, to the mechanical effect of the tamponade plus the inhibitory action of the lactic acid upon the saprophytic microorganisms in the nose, as he obtained similar results by the use of lactic acid tamponade alone.

Possibilities for permanent relief of the distressing symptoms are based on one principle, and that is the narrowing of the intranasal space. This may be accomplished by either of two methods: *1.* Approximation of the lateral wall of the nose in whole or in part to the septum; *2.* Submucous implantation of various substances into the septum with the idea of thickening the latter and thus lessening the intranasal space. Lautenschlager, in 1916, was the first to employ the method of approximating the lateral wall to the septum. His operation, in short, is as follows: The incision is the same as that employed in a classical Caldwell-Luc operation. A large piece of bone is removed from the facial maxillary wall and is preserved for further use. The mucus membrane of the antrum—healthy or diseased—is scarified completely. The nasoantral wall is then chiseled at its floor and after infracting this wall in several places, it is pushed over to the septum. The preserved piece of facioantral wall is then introduced subperiosteally into the medial surface of the nasoantral wall. The septum is scarified with the hope of creating synechia and the antrum firmly packed to hold the lateral wall in apposition to the septum. Several modifications of this original procedure have since been published, among which are those of Halle, Hinsberg, Wittmaack and Von Borries. These operative modifications have the same aim in view, *viz.*, approximation of the lateral wall to the septum. Following the publications of Lautenschlager and Halle, I followed their procedures in a fair number of cases, but was finally obliged to discontinue them for the following reasons: The operations are formidable, very extensive and radical; they necessitate prolonged hospitalization and postoperative care; occasionally severe complications arise, *viz.*, sinusitis and dacrocystitis and a persistent antral fistula; the end results are usually disappointing. In most instances the lateral walls receded to their normal position and the synechiae become stretched and few in number, so that the nasal passages are practically in the

same condition as before operation. In those few cases where the adhesions are intact, while it is true that secretion and crusting have diminished considerably, there is, nevertheless, great difficulty in expelling the crust and secretions which continue to be present. My observations are taken not only from my own experience, but largely from the results of European rhinologists whose work I saw while abroad several years ago. There I had the opportunity of examining many of these patients in various postoperative stages, varying from the time of operation to several years after. At the recent Academy meeting in Boston, Dr. Wachsberger presented his modification which seemed to me to be perhaps the best of the radical type of operation. I have had no personal experience with the procedure, but from its description would regard it as being less productive of complications and less formidable of performance.

Even before the Halle or Lautenschlager methods were presented, my former associate, Dr. Joseph Beck, and I, in 1912, conceived the idea of forcing the septum toward the lateral wall of the nose. We attempted to do this by means of intraseptal implants, placing them under the mucoperichondrium and thus bringing the outer septal wall toward the lateral nasal wall. In our first attempts inlays of paraffin were used, but within a comparatively short time these sloughed out. We then decided to try a homogeneous substance and began to employ fascia lata. This healed in very nicely, but after a short period the implanted fascia lata contracted to such an extent that scarcely any narrowing of the space was discernible. The next material used, while very successful, was not always easily obtainable, *viz.*, bone and cartilage from a previously operated septum resection. We found that it required two septi, as a rule, to fill up one side of an atrophic rhinitis space and often it was difficult to have all three patients on hand at the same time. When such fortunate circumstances did occur, our results were more than gratifying, as in nearly every instance a physiological cure resulted. Quite often, too much shrinkage and absorption occurred after a year, so that the patients were not as free from the symptoms as we had anticipated. We usually waited some six months before operating on the second side for fear of losing the nutrition of the septal cartilage, as occurred in one case with a resulting perforation. Therefore, we attempted to use cartilage or bone

from the patient himself, cartilage from the rib or bone from the tibia. As occurs occasionally in all plastic, intranasal surgery, infection sometimes took place and in those cases we lost our transplant, following which it was sometimes difficult to persuade the patient to submit to another operation.

Having obtained good results in the use of ivory implants in plastic surgery about the head, we conceived the idea of using this material for our intraseptal implants. Not having previously heard of anyone using this material in intranasal surgery, I was obliged to work out my own methods as to size and shape, after obtaining the implants by the rather primitive method of sawing the pieces from the tusk of an elephant. The shape of the transplant, amount used, and the placing of same was a matter of experience to be worked out according to individual needs. In my early attempts the amount used was too great and I failed to round off the sharp corners, so that in two instances the ivory implants sloughed out. Such accidents are now entirely avoidable, as the material can now be obtained in various sizes from the supply house of V. Mueller & Company, of Chicago. However, a complete outfit of saws, files, vises, etc., should be available for use in occasional instances. Later I modified the preparation of the ivory by perforating it with the idea that the resultant connective tissue would grow through the openings and while holding the implant in place, would also give nourishment to the underlying cartilage. Since employing this modification I have never had an occurrence of sloughing and consequently feel that the theory was justified. Inasmuch as I am of the opinion that every case of ozena is associated with chronic ethmoiditis and in many instances associated, in addition, with chronic suppuration of the other sinuses, especially the antrum, attention must be given the sinuses and every effort, surgical or otherwise, made to eradicate the underlying pathology before attempting intranasal implantation. In every case a thorough exenteration of the ethmoid cells is done, the middle turbinate allowed to remain since it is advisable to preserve as much intranasal tissue as is possible. Following the sinus operation it is advocated to wait at least six weeks before the intraseptal implantation is done; in the event that conditions do not justify further procedure at the end of this period a further wait is, of course, necessitated. A number of small perforated ivory implants are prepared, ranging from

one-half to one and a half inches in length, from one-quarter to one-half inches in height and from one-eighth to one-quarter inches in thickness. All sharp edges must be well rounded. Since no shrinkage occurs in this material and all of these operations are done under local anesthesia, with its resultant temporary retraction of the inferior turbinate, one should guard against a tendency to employ too thick implants. As stated before, in my earlier cases this occurred and when the turbinate returned to its normal condition on the following day, I discovered that the space was too narrow for normal breathing. Thereafter I was very careful to avoid the use of too large implants and consequently that objection was easily overcome. The implants are sterilized by boiling, which in no way injures or roughens them. A small incision is made in the mucocutaneous border and the mucoperichondrium is carefully dissected as in an ordinary submucous resection. Special care must be taken to preserve the entire flap, for if a tear occurs at any point there is great likelihood of infection resulting, necessitating the removal of the implant. Every effort must be made to dissect as close to the floor as possible, for the greatest amount of atrophy usually occurs at this point. The implants are then slipped through the opening and as this is in the skin, there is but little danger of tearing the flap. From one to three implants are employed on either side, depending upon the individual requirement. It is not necessary to sew the wound, but this may be done if preferred. I place a small Bernay's splint over the incision and remove it on the following day. One is astonished at the slight amount of reaction ensuing, for upon inspection of the nose during the next few days, one sees only a slightly reddened mucosa with no edema or exudate. In my earlier experience, when only one side was operated at a time, an improvement of the operated side was noted within 24 hours, as there was but little secretion and no crusting or odor, while the unoperated side presented the usual picture. The one symptom of which the patient complained was stuffiness, not any real obstruction, but comparatively, as the operated side was now narrow while the other was still enlarged. The improvement continued with a gradual diminution of the secretion so that in a few weeks a physiological cure resulted.

My ten years' personal experience with ivory implantation has been very extensive and I wish to emphasize that in only

a small percentage of cases does the implant extrude itself and when this does occur, it is shortly after insertion. In only one case in my experience did a fistula result. The majority of patients operated by this method obtain a physiological cure and the remainder are greatly benefited. In my early employment of this method I had occasion to remove several implants found to be too large and in each case they were fully encapsulated, the capsules being thick and firm. I have a number of slides which demonstrate the ivory implant in position with narrowing of the nasal cavities and in some cases apparent hypertrophy of the inferior turbinates. Some of these slides represent cases operated as early as ten years ago and to date none of the implants have come out. Once firmly embedded they remain indefinitely.

There is no question but that the surgical treatment of ozena is the proper one, as palliative measures absolutely fail to give permanent results. As to what surgical procedure be employed, this depends on the individual operator, but as in any undertaking, the procedure which accomplishes the desired results by the simplest means and with the least amount of danger is the method of choice.

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2561 N. Clark Street.

THE RATIONALE OF SURGICAL TREATMENT FOR OZENA. PRESENTATION OF CASES OPERATED BY THE AUTHOR'S METHOD.*

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One of the reasons for the rare employment of surgical treatment in ozena is the failure to understand the rationale that underlies its use. The surgery alluded to refers only to those operations which narrow the nasal passages, such as the classical operation of Lautenschlager and its modifications.

The unusual width of the nasal chamber, whether it is primary to the pathological process or vice-versa, is present in all cases and it can be said that the wider the nose is the more severe are the symptoms. One has to recollect only the cases with apparently unilateral ozena and deviation of the septum to the good side to measure the truth of the above statement. The word "apparently" is used because ozenous disposition seems to be contained in the mucosa of both sides. Crusts will soon form in the narrow side if it is widened by a submucous resection of the septum. This proves that there is a definite relationship between the width of the nasal air passage and the atrophy, crust formation and fetor.

What is this relationship? The normal change from negative to positive air pressure in the nose with inhalation and exhalation is known as an important factor in sinus ventilation. Its influence on the blood circulation, however, has not been appreciated. We know of the circulatory changes in the heart and the great vessels in the chest effected by inhalation and exhalation, and of the hyperemia observed in the nasal mucosa brought about by the creation of a vacuum with mass suction, and can safely assume similar happenings to a lesser degree as a result of nasal breathing. The resistance to respiratory pressure when measured is much greater in the nose than in the mouth of an individual with a normal nose.

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Not so with ozena patients, where the resistance in the nose is practically the same as in the mouth due to the anomalous width of the nasal passages. The beneficial effect of the normal respiratory pressure change upon the blood circulation of the nasal mucosa is therefore lost in ozena. This does not only promote the atrophy, but it also reduces the secretory activity of the nasal glands. The result is the secretion of a scanty amount of mucus of high viscosity, which when exposed to the evaporating influence of the increased amount of air which passes through the unusually wide nares, dries to form crusts.

The aforegoing remarks stress the physical factor in the etiology of ozena. To hold these solely responsible for the production of ozena is neither attempted nor assumed by the writer. They help to understand the results that are achieved by the physical changes that are brought about when the nasal passages are narrowed surgically.

When the lateral nasal wall is transposed medially toward the septum or material is implanted under the mucoperiosteum, a smaller amount of air will pass over a relatively larger surface. Evaporation of the nasal secretion is thus diminished and the alternating respiratory pressure effected by inhalation and exhalation is restored. This in turn has a favorable influence upon the blood circulation and on the amount and quality of the secretion from the mucous glands. The blood circulation is further aided by the formation of new anastomoses through the adhesions between the septum and the turbinates. Last, but not least, the patient is again able to clean the nose by blowing.

A detailed description of the author's modification of the Lautenschlager operation—which was developed for greater efficiency, simplification of the technique and to make it a minor procedure in the eyes of the patient—was presented before the Annual Meeting of the American Academy of Ophthalmology and Otolaryngology in Boston in September, 1933.

The writer has operated on more than 50 cases, using Halle's Modification, and on 14 cases with his own technique. The end results of these latter cases have been observed for as long as five years. The crust formation has ceased in all of these; occasionally some soft scabs are seen in the nasopharynx. The atrophy has been improved and the fetor eliminated in all cases.

The following are the histories of three ozena patients who are presented here tonight:

Case 1: O. J., male, Porto Rican, age 18 years. First seen Nov. 17, 1928. Complained of purulent nasal discharge since the age of 6 years. Crusts and foul odor from the nose. Two sisters have similar complaints. Tonsils and adenoids were removed one year ago without benefit to the nasal condition.

Examination: The right inferior turbinate was reduced in size to a narrow ridge. Left inferior turbinate was absent. The middle turbinates were relatively large and bullous. Both nares were filled with dry greenish crusts. Marked fetor. Transillumination: Both frontals and both antra were dark. X-ray examination revealed the absence of both frontals. The antra were very small, but contained no exudate. The left antrum was smaller than the right one. Irrigation of the right antrum yielded a clear return flow.

Diagnosis: Ozena. Dec. 4, 1928: Modified ozena operation on the right side. Recovery uneventful. Right antrum packed twice, the packing having been left *in situ* for five days each time.

Dec. 27: The opening into the antrum is closed. Marked congestion in the right nares with numerous small mucous polyps in the middle meatus.

Jan. 8, 1929: Right nares hyperemic and moist. No crusts. Left side is lined with crusts. Advise operation for the left side.

July 30: Modified ozena operation on the left side. The antrum is found to be greatly reduced in size, about as large as a bean situated posteriorly and superiorly in the maxilla.

Dec. 7, 1930: Right nares clear and hyperemic. Soft scabs in the nasopharynx on the left side.

Oct. 31, 1933: Mucosa congested and moist. No crusts on either side. Soft scab in left nasopharynx. Patient states that he never irrigates the nose. Recently married. No odor noticed by his wife or himself. Subjective sense of smell: fair. Had no sense of smell before operation.

Case 2: C. A., female, age 20 years. First seen May 15, 1930, and complained of purulent and crusty nasal discharge for seven years. Frequent frontal headaches. Had meningitis at age 7 years. Left ear discharging for ten years.

Examination: Both nares are lined with greenish, dry and fetid crusts. All turbinates are atrophic and the nasal lumen is unusually wide. The nasopharynx is filled with crusts. Transillumination: both antra are dark. Irrigation of both antra yields clear return flow. X-ray examination shows all sinus walls to be thickened. There is no exudate in any of the sinuses. Wassermann reaction is negative.

Dec. 23, 1930: Bilateral modified ozena operation. Antra were packed twice during the postoperative period.

March 3, 1931: Mucosa slightly hypertrophic. No crusts. No fetor. No headaches.

Nov. 1, 1933: Patient does not irrigate. Feels well. Breathes well through the nose since operation. No odor is noticed either by her husband or herself. Subjective sense of smell is fair. Extensive adhesions. Mucosa is congested and moist. There are no crusts except for a small soft scab in the nasopharynx.

Case 3: B. M., male, Jewish, age 14 years. First seen Feb. 15, 1933. Nine years ago his teacher noticed a bad odor. He was placed by himself apart from the other pupils. Had purulent and crusty discharge from the nose. Irrigated nose daily. Mother and two sisters and one brother have similar complaints. They were all examined and showed various degrees of atrophic rhinitis.

Examination: Both nares lined with dry, greenish, ill-smelling crusts. Both inferior turbinates are atrophic. The middle turbinates are apparently enlarged. Transillumination: All sinuses are dark. X-ray examination shows the absence of the frontal sinuses. The ethmoids are cloudy. Both antra show sclerotic walls, are small in size and contain no exudate.

Diagnosis: Ozena. April 3, 1933. Modified ozena operation on the left side under general anesthesia. Recovery uneventful. The antrum packed twice during the postoperative care.

Nov. 1 (last examination): Left antrum: mucosa is moist and congested. No crusts on that side. There are no adhesions between the left turbinate and septum, yet the lateral wall is in the transposed position close to the septum. The right nares and nasopharynx are filled with greenish malodorous crusts. Advise operation for the right side.

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SYSTEMIC COMPLICATIONS IN SINUSITIS.*

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When Billings, in 1911, expounded his theory of focal infection, a new era was opened in the practice of clinical medicine. Age-old riddles were solved, light was cast into the darkest recess of pathological obscurity and it seemed as if, at last, we had found the underlying factors which are responsible for many hitherto obscure diseases.

In our enthusiasm we attributed too much to these infective foci, and we also deceived ourselves into thinking we found infective foci where none existed. After two decades the pendulum is swinging the other way. Many are beginning to believe that we must still seek elsewhere for most of the etiological agents we so recently felt sure we had run to earth.

As is usually the case, the truth probably lies between the two extremes. As regards the accessory sinuses of the nose, this is undoubtedly the situation. Not only does this hold good for the focal infection theory, but also for the theory of allergy or protein sensitization, which has received almost as much attention as that first named. We have been taught that certain forms of sinusitis are due to allergy, and conversely that certain individuals are sensitized from sinus infections. Thus we have the situation of diseased sinuses acting as foci of infection; of sinus disease arising because of infection from a focus elsewhere in the body; of diseased sinuses producing proteins which sensitize their unfortunate possessor to allergic conditions in other parts of the respiratory tract, and finally, of sinus disease which is due to sensitization from an outside protein.

Therefore, in a consideration of systemic complications in sinusitis, we must take all these viewpoints into account and harmonize them if possible. No one man's experience can

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be reasonably expected to include all types of affections in their multiple manifestations, neither can the findings of a single individual be expected to parallel exactly those of any other. Therefore, if I seem to dogmatize, it will be because amid such conflicting opinion one will become confused unless he keeps his goal very clearly before him.

Whether we look upon sinus disease as a product of metastatic invasion or regard it as the underlying cause of pathologic conditions elsewhere in the body, there is no question that the complications which arise during the course of sinus disease have, as their underlying etiologic factor, a previous bacterial invasion of some one of the accessory nasal sinuses. Therefore, anything which was instrumental in the original introduction of the infection has been instrumental in producing the complication.

The anatomical structure and physical position of the offending sinus may be responsible for the distribution of an infection by direct extension, quite as much as by pouring its diseased content into the blood stream. Located at the very top of the respiratory and digestive tracts, there is ample opportunity for such direct communication. Fenton,[1] addressing the American Laryngological Association last year, declared that absorption through the sinus mucous membrane is "relatively slight, and opinion now tends to minimize the sinuses as sources of focal infection. Rather, they may act as reservoirs of infective material which passes down into the respiratory and digestive tracts and is elsewhere absorbed."

Acting as a reservoir means that a dam of some sort exists to prevent the normal evacuation of the sinus secretions. If such obstruction occurs, it is easy to see how the close anatomical relations borne by the sinuses to the brain and the other important structures of the head may bring about most serious complications should the pressure of the dammed up secretions become so great as to cause rupture of the fragile bony walls of these small cavities. The floor of the anterior cranial fossa often serves also as a roof to the ethmoid and frontal sinuses and frequently will be found to be as thin as a sheet of paper. With such pressure as complete retention of the secretion of an infected mucosa will soon produce, there is little to hinder the rupture of such an inadequate partition,

which will be followed by seepage of these secretions into the overlying dura and the probable production of intradural abscess. If the walls of the sphenoids are equally thin, a similar accident will permit the flow of septic material into the middle cranial fossa, with almost certain sequelae of brain abscess or meningitis.

A word of caution might well be interpolated here as to the possibilities of sinus disease co-existing, although entirely independently, with such brain lesions as cerebellar tumor or affections such as encephalitis lethargica. Lobell,[2] in 1926, reported three such cases.

If the maxillary sinuses are diseased, their relative remoteness from the brain does away with any likelihood of intracranial complications, but their power to initiate infections which may extend to the oral cavity, the throat and the lungs is all the more formidable. A great deal of work along this particular line has been done by Mullin,[3] of Crile's Cleveland Clinic. By an extended series of experiments, he demonstrated a lymphatic route of extension from maxillary antrum to lung, which, he thinks, functions in the development of the bronchial condition as a secondary lesion from the original infection located in the antrum. Even when there is but little secretion being given off from the antrum into the nose, he is still of the opinion that the infection may pass by way of the lymphatics. With a nose full of pus, inhalation is a more probable path because some of his experiments "illustrate strikingly the readiness with which substances constantly present in the upper air passages reach the lungs by inhalation, and we cannot wisely ignore this fact in trying to interpret the relation between infection of the upper and lower respiratory apparatus."

If clearing out the offending sinuses does not result in immediate cure of the lung condition, it is by no means a positive proof that the antral infection was not responsible for the bronchial one, because, once the bronchial condition is well established, removal of the original causal factor cannot be expected to affect it in any way. The proper measures, such as postural drainage, injection of autogenous vaccines, or climatic treatment, must be resorted to, no matter what was the origin of the bronchial condition. But when the sinus disease has existed for some time and lung involve-

ment is still in its early stages, proper treatment of the diseased antra will serve to halt the advance of the lung condition, so that it often subsides spontaneously.

The old-fashioned "winter coughs" suffered by the generations past, which modern clinicians usually attribute to pulmonary tuberculosis on the strength of the reports handed down from the practitioners of that bygone day, were more probably instances of bronchiectasis which had their origin in sinus infection. In dispensary practice today it is common to see children who have undergone one or several of the exanthemata, particularly measles and whooping cough, and thereafter have retained a "chronic cough" with a general syndrome of chronic toxicity. A thorough physical examination reveals bronchiectasis with disease of one or many sinuses.

Frequently such children will be sent to preventoriums, or will be diagnosed as frankly tuberculous and placed under treatment for that condition. Such diagnostic errors are not wholly bad, because the hygienic measures suitable for the one condition are likewise excellent in the other. But as the sinus disease seldom rights itself, the child is left untreated so far as this lesion is concerned and no real progress toward permanent recovery can be expected.

Even with all that has been said and written concerning sinus infection, its likelihood to become a distributing point whence toxins are carried to remote parts of the body and its possible effect upon the general health and well being, it is my opinion that the prevalence of this condition is not yet fully recognized. The average acute attack of sinusitis undergoes spontaneous resolution and goes little regarded — just a cold. But let some anatomical or bacterial "accident" make its appearance and at once we have a set of conditions which may lead promptly to something very grave indeed.

If we have *1.* any anatomical structures sufficiently abnormal as to obstruct free drainage, *2.* an infection of greater virulence than ordinary or *3.* any factors lessening the subject's resistance, then spontaneous resolution cannot take place. The nose has its natural defenses — bactericidal properties of its normal secretions and an ability exercised by the ciliated epithelium which expels invading bacteria or other foreign material before it has opportunity to do harm. Yet

any of the factors mentioned above may easily overcome these natural powers of defense, and infection quickly finds a congenial lodgment in one or several of the sinuses. This can be quickly redistributed by absorption into either the blood or lymph streams, or it may — as we have already seen — travel by gravity or direct continuity to what seem, at first thought, to be very remote areas.

Often these infections are highly insidious. They cause no pain; there is only occasionally any discharge; there is no cough, headache, or any of the unpleasant manifestations commonly associated with disease of the sinuses. Yet the patient goes steadily downhill. The extensive mucous lining of the paranasal region gives a wide surface for absorption; the low grade infection continues and eventually a whole family of ills, ranging from colonic stasis through arthritis to nephritis or endocarditis, is prone to arise.

Such complications can take place even when there is no actual necrosis of the bony walls of the sinus. If a virulent organism gets opportunity to enter, an acute inflammation may be superimposed upon a chronic one of long standing; what was but a partial blockade develops into complete stoppage of all drainage; the pressure of purulent fluid eats away the thin partitions so that rupture takes place, or even without actual rupture, seepage becomes possible. With a strong and intact bony wall such retention may prove an active source of general systemic infection, because inflammation of the mucosa may be accompanied by a thrombophlebitis which quickly extends to the veins which communicate with surrounding structures, such as the orbit. This gives such an acute condition as orbital cellulitis or even a more generalized osteomyelitis.

The intimate relationship between the paranasal sinuses and the brain gives ground for the prevalent conviction that sinus disease often produces psychoses or even permanent grave mental imbalance. Watson-Williams,[4] who has given especial attention to this relationship, points out that not alone direct continuity may be responsible for injury to the nerve structures, but the same mechanism which operates in the production of the joint toxins responsible for arthritis may be invoked "to explain the particularly frequent relationship of nasal sepsis to disturbances of mind and character; and

also, why the mental outlook, as well as the physical disabilities, improves so quickly as a result of operative removal of the source of sepsis. A constitutional failure of the organism to form antibodies or biochemical susceptibility to toxins peculiar to the individual may determine mental unsoundness following infection and explain the restoration of the mentality to a normal state as the result of removing the sources of infection." And he concludes with this significant paragraph: "The slighter mental disturbances amounting to alteration in character, disposition and power of concentration are fruitful causes of unhappiness in the home, of broken hearts, of business losses, and loss of time and energy to the working man; thus they are really far more important than they might appear at first sight, and by no means to be lightly treated."

It might be added that such mental disturbance may likewise be dependent, not directly on the toxic effect of intranasal sinus infections upon the nervous system, but upon its more remote relations with vital processes, such as digestion, respiration and the circulation of the blood. The action of the heart, for example, is stimulated or inhibited by strong stimuli exerted upon the nasal structures. We revive a person who has fainted by using smelling-salts; but faintness, or even complete unconsciousness, may be brought about by the odor of blood or even an intrinsically pleasant odor like that of lilies of the valley. Therefore, the nerve supply of the nasal structures must have an intimate relation with that controlling the heart.

There is, indeed, direct anatomical evidence, brought forward by Cajal and other histologists, explaining the influence which nasal innervation has upon the heart when in perfect health. This should also account for the interference with heart action which is so often seen in patients suffering from nasal sinus disease, in whom no actual cardiac lesion can be demonstrated. It has been observed that when such patients receive proper treatment for their sinus trouble, the heart difficulty quiets down and usually disappears entirely.

That the sinuses may act as foci of infection contributing to the institution of grave cardiac lesions has been recognized for many years. As far back as 1924, Miller[5] made extensive examination of the nose and throat conditions in cadavers, where death had been caused by conditions apparently uncon-

nected with these portions of the body. He published an account of three such necropsies, where the direct cause of death had been disease of the heart, but there was a post-mortem finding of maxillary sinus disease which had every appearance of having antedated the heart complication. Yet none of these patients had given a history in any way suggesting antral involvement, and the routine examination of the head had reported the sinuses negative. No Roentgenographic investigation was made, but this is hardly to be expected when there was no history nor active symptoms to indicate that the extra trouble and expense involved in the X-ray examination would be justified.

A distinction is made by Watson-Williams between "a focal reaction to infection" and "focal sepsis." The focal reaction, according to this distinguished British otolaryngologist, is an incident; the essential feature of focal sepsis is the continued activity of the infecting organisms and (unless potentially a source of toxemia) a focal reaction is not focal sepsis, but the result of focal sepsis. These focal reactions are "the pathological event of a *bacterial* invasion." They correspond, he believes, with the process which Adami once described as *subinfection*, ". . . that is to say, the bacteria thus carried in do not multiply . . . they are destroyed, but with their destruction the liberation of their toxins causes a poisoning of the cells immediately around them, and the accumulative action of these toxins, whether locally or at a distance, brings about the death of these cells and replacement by fibrous tissue."

This etiological explanation applies not only to cardiac lesions, but to arthritis and similar pathologic conditions now universally conceded to be produced by focal infection. Conversely, it may also offer an explanation of chronic hyperplastic sinusitis, which, often accused of being the underlying factor in various widely differing diseases, may itself be due to a focal infection from some other part of the body. The latest theories in regard to this were recently summed up by Grove and Cooke[6] as: *a.* allergy, in the skin-sensitive type of patient; *b.* infection; and *c.* a combination of allergy and infection. These authors themselves, however, believe that the hyperplasia is due to a primary bacterial infection in a person with an allergic constitution and bring forward proof, both clinical and experimental, to reinforce their point of view.

Retrobulbar optic neuritis, while not infrequently associated with suppurative conditions in the sinuses, notably the sphenoids or posterior ethmoids, is also thought to originate from a nonsuppurative hyperplastic inflammatory process. These views do not find universal acceptance, but no less an authority than Sluder has accounted for optic neuritis by the extension of a hyperplastic process from either sphenoids or ethmoids to the channel carrying the optic nerve. This causes such a decrease in the lumen of this canal that the nerve is compressed and hindered in its function, if indeed, it is not irreparably damaged. As the optic nerve is particularly susceptible to toxins, such complications may also arise when the sinuses are acting as foci of infection and elaborating toxic material which is conveyed to the nerve through the medium of the blood stream. It may also, especially if it be the ethmoid labyrinth which is in question, attack the optic nerve directly at the point where labyrinth and nerve come into intimate contact at the posterior external angle of the last cell (Loeb).

The close connection between eye disturbances and sinus disease is quite widely recognized, but the exact mechanism by which this is accomplished is still a matter of dispute, even among the most well informed. It is generally conceded, however, that hyperplasia of the mucous lining of the sinus makes it more vulnerable to infection, even though the existing condition may be the result of infection earlier incurred.

SUMMARY.

Focal infection and protein sensitization have both been cited as operative in the production of various diseases believed to be dependent upon pathological conditions in the paranasal sinuses.

Though considerable difference of opinion is now prevalent, there is very general agreement that all the complications of sinus disease must have, as their underlying factor, a previous bacterial invasion of the offending sinus.

Because of their anatomical location in close relation to the brain, disease in the paranasal sinuses may easily be communicated to the brain, either by direct extension through rupture or seepage because of the thinness and vulnerability of the bony wall, or by conveyance through the blood stream or the lymphatic channels.

Inhalation may play in the conveyance of sinus — notably antral — infection to the lungs. There is reliable clinical proof that focal infection originating in the sinuses is responsible for many cardiac lesions.

The connection between such autointoxication and disturbance of the mental state, amounting frequently to actual psychoses, has been quite satisfactorily established, both in this country and abroad. The optic nerve is especially susceptible, which makes sinus disease accountable for many cases of retrobulbar neuritis or even optic atrophy, though this last is more often produced by pressure from hyperplastic but nonsuppurating mucosal lining of the offending sinus.

The part played by sinus disease in general medicine is a very important one, and an intelligent understanding of the anatomical and pathological conditions of this region is imperative to the proper practice of medicine.

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10 W. 82nd Street.

1.—SQUAMOUS CELL CARCINOMA OF THE UVULA, SOFT PALATE AND TONSILS.

2.—ACUTE MASTOIDITIS, LATERAL SINUS THROMBOSIS, ABSCESS OF INTERNAL JUGULAR VEIN, OPERATION AND RECOVERY.*

DR. CARL KAPLAN, Brooklyn.

The first case is that of a male, J. B., age 73 years, retired manufacturer. His present illness can be traced to Jan. 1, 1931. His main complaint at that time was irritation of the throat, which produced inability to sleep on account of a slight cough. He consulted a family physician, who prescribed a gargle. Having no relief from this medication, he went to a second physician, who treated him for several weeks without obtaining any relief. In the latter part of 1931, this patient consulted his dentist, who, during the course of his treatments and examinations, discovered that his whole throat, including the soft palate and part of the hard palate, were constantly inflamed, despite the many gargles and medication the patient used. The dentist insisted that he consult a laryngologist, and the patient came to my office the latter part of June, 1932. His main complaint at that time was slight soreness of the throat; there was no dysphagia or cough or loss of weight.

Past History: About 30 years ago, patient was troubled very frequently with acute follicular tonsillitis, which was complicated with peritonsillar abscesses. He had at least four attacks of peritonsillar abscesses, which were incised. For the past 25 years, however, the patient had no throat trouble or any nasal obstruction.

Habits: Appetite good, no distress after eating, bowels regular, tobacco none, alcohol none.

Pulmonary Symptoms: No cough, no bloody expectorations.

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Cardiac Symptoms: No shortness of breath, no vertigo.

Genito: Urinary symptoms negative.

Surgical History: Four incisions for peritonsillar abscess.

Family History: Father died at 60 years of nephritis. Mother died at 40 years, cause unknown. No history of cancer in the family. *Physical Examination:* Patient appears to be well nourished and well preserved for his age, of good spirits, does not appear cachetic. Head: Eyes: Pupils equal, react to light and accommodation. Nose negative. Ears negative. Mouth: All teeth absent and replaced by two artificial dentures. The mucous membrane covering the soft palate and that part of the hard palate beyond the contact point of the superior artificial denture was red and inflamed. The anterior pillar of both faucial fossae, tonsils and posterior pillars were of the same red color. The pharyngeal wall was also slightly injected; the nasopharyngeal space examined with a laryngeal mirror, was also somewhat injected. The larynx was negative. Neck: There was a slight enlargement of the gland at the angle of the jaw, and also of the submaxillary gland. Chest: Lungs were normal on palpation, percussion and auscultation. Heart: No murmurs heard at the apex, or at the right or left second intercostal space. Abdomen negative. Extremities negative.

On my first examination, my impression was that the patient was suffering from a low type of infection of the throat, provided that lues, tuberculosis and malignancy could be excluded.

Since primary tuberculosis of the palate and tonsils are so rare, it was ruled out at once. Lues and malignancy were seriously considered. On the second visit, I took some blood for a Wassermann test. This came back negative.

In the meantime, the throat condition remained the same.

During the following two weeks, the patient was under close observation. At the end of July, I examined his throat once again, this time with a large magnifying glass. I was then able to visualize very small ulcers around the left margin of the uvula and tip. There was also a slight ulceration of the left tonsil near the superior pole. Several pieces of tissue for biopsy were removed from both tonsils, soft palate and

uvula. These were sent to the pathological laboratory at the Jewish Hospital. On July 29, 1932, Dr. Lederer, Pathologist, reported the following: Several pieces of grey tissue discolored pink with mercurochrome. Microscopic examination shows squamous celled carcinoma, Grade III.

The proper course of treatment was very important. The choice lay between surgical or radiotherapy. In order to do a radical surgical operation in this case, it would have been necessary to remove the entire soft palate, both tonsils and both anterior and posterior pillars, together with a complete block resection, removal of the cervical glands of the neck.

Considering the age of the patient, the complications following such a radical surgical procedure were very serious. The radio therapist, Dr. Louis Nathanson, and myself decided that it was for the best interest of the patient to treat him with radium and deep X-ray therapy.

On Aug. 4, six 1.065 millicuries of radium were inserted; two in the anterior pillar of the left tonsil, two into the posterior pillar of left tonsil and two in the tonsil itself. The reaction after this treatment was very slight. Deep X-ray therapy was immediately started.

On Aug. 18, six more implants, 1.0 millicurie, were inserted as follows: One into the right side of soft palate, one into left side of soft palate, one in right posterior pillar, one in the right anterior pillar, one inserted into the left posterior pillar and one in the uvula. The reaction following this radium treatment was very slight.

Until Oct. 15, the patient was treated with deep X-ray therapy, receiving the maximum dose that could be applied.

On Oct. 20, examination of the mouth revealed that the mucous membrane was assuming a normal color. The ulceration around the uvula and soft palate were practically healed. There was a mass of glands palpable at the left submaxillary region. The gland in the region of the left angle of the jaw was still present. It was decided to use more radon seeds, to be implanted into these glands.

On Oct. 22, seven radon seeds, each one containing 2.0 millicuries of radon, were inserted into the left submaxillary glands and at the angle of the jaw. These were inserted with-

out any anesthesia. The reaction following this treatment was very slight. Patient complained of dryness of the mouth and slight loss of taste.

Examination (Nov. 1): Slight injection of the mucous membrane of hard and soft palate and both tonsils. The left submaxillary glands are much smaller in size. Patient has not lost any weight, his spirits are excellent.

The second case I am presenting illustrates an interesting complication. J. D., age 17 years, born in United States, single, high school boy, came to my office on June 30, 1932. Present illness dates back four weeks. At that time, he had an upper respiratory infection. One week later complained of pain in right ear, and a myringotomy was performed on the right side by his family physician. The ear drained for three days. It was incised a second time, by his family physician. For the past two weeks, patient has had intermittent chills, accompanied with high temperature, and sweats.

According to the patient's own statement, these chills occurred about 7 P.M., and lasted about 20 minutes. This was followed by a very high temperature and profuse sweating; the patient felt well again until the following night, when the chill, fever and sweat recurred.

Past History: Did not have any serious illness previous to this one.

Surgical History: Had tonsils and adenoids removed two years ago.

Family History: Father and mother alive and well.

Physical Examination: Nose: Small amount of pus in right nasal fossae. Throat: Evidence of previous tonsillectomy. Eyes: Normal, react to light and accommodation. No nystagmus, no petechiae of the conjunctiva. Neck: No rigidity, no glandular enlargement, no tenderness over the course of the right jugular vein. Chest: Clear; heart, no enlargement. First sound is harsh, sounds are rapid, regular and of good quality. Abdomen: No palpable organs, no tenderness. Extremities: No deformities, knee jerks normal, no inguinal, axillary, adenopathy. Transillumination of sinuses: Normal antra and frontals. Left Ear: Tympanic membrane normal. Right Ear: External auditory canal slightly injected. Small

perforation in the posterior inferior quadrant. Slight sagging of the posterior superior canal wall. Hearing Test: Right ear diminution of hearing there was slight tenderness over the right mastoid tip. Temperature 104°. Pulse 120 and respiration 20. Diagnosis: My impression at the office was that the boy was suffering from Acute Mastoiditis, complicated by a right lateral Sinus Thrombosis or a right primary Jugular Bulb Thrombosis.

Patient's mastoids were X-rayed on the same day. The report is as follows: The left mastoid is well developed. It is of the pneumatic type and presents normal clarity. The trabeculations appear sharply defined and intact.

The right mastoid shows a generalized haze. Accentuation of the basal triangle is noted, indicative of heavy infiltration in the antral and periantral region. The cellular markings as compared with the opposite side appear in most part destroyed. Thinning out and lack of clarity and distinct appearance of the destructive process. The pathology is noted in the tegmen and zygomatic regions, extending to and including the tip. The latter is markedly involved as the former.

The anterior sinus wall is observed, appears sharply demarcated and the above findings indicate a destructive mastoid process on the right.

Diagnosis: Right mastoiditis. Patient was admitted to the Jewish Hospital on June 30, at 9:30 P.M. On admission, his chief complaint was chills and high fever. Patient had a chill upon being placed in bed, which lasted 20 minutes. After the chill the temperature rose to 106.6°, and he complained of headache.

A blood culture and complete blood examination and urinalysis were ordered. The report of the blood examination was as follows: Red blood cells, 3,050,000; hemoglobin, 55 per cent; white blood cells, 13,200; polys, 82; mononuclear and transitionals, two; lymphocytes, 16. Urine: Specific gravity, 1012. Reaction acid, albumin and sugar negative. Microscopic: Negative.

On July 1, at 9:00 A.M., patient was taken to the operating room. Nitrous oxide and ether were administered. A postauricular incision was made. Cortex was removed, cells were

found to be diploic in character. The entire mastoid tip and tegmen plate were soft and necrotic. There was present a perisinus abscess. The sinus plate was soft and necrotic. This sinus plate was removed from the region near the jugular bulb posteriorly within about one-half inch from the torcular end. The sinus wall appeared dark. A nick was made near the torcular end and a flow of free blood was obtained. This was controlled by a sinus plug. Another nick was made in the sinus near the jugular bulb. Free blood was obtained, which was also controlled by a sinus plug. The sinus wall was incised again between the aforementioned places, no clots were present, bleeding controlled by the additional packing with three sinus plugs.

Following this procedure, an incision was made along the anterior border of the right sternocleidomastoid muscle for the purpose of ligating the right internal jugular vein. After the carotid sheath was opened, the internal jugular vein was identified, freed from the vagus nerve and carotid artery. The vein was freed low down and high up in the neck.

Both the right internal jugular and facial veins were ligated with double black silk sutures. The ligature on the facial vein was cut short, the ligature on the internal jugular vein was made long and carried out into the neck wound, which was packed with iodoform gauze. The mastoid wound was dressed and patient was returned to bed in good condition.

One thousand cc. of a 5 per cent Glucose Solution was given intravenously, immediately patient reacted well. That evening, temperature at 8 o'clock was 99°. Took fluids very well.

His condition continued to improve until the evening of July 4. Temperature rose to 104.5°; the following morning it dropped to 100° and remained between 100° and 100.5° until the evening of July 7. Temperature rose to 103.5°. He also complained of chilly sensation, but had no distinct chill. In addition to the chilly sensation, he complained of pain in right side of neck with slight dysphagia. The following day patient was taken to the operating room for a sinus dressing. The sinus was inspected, free blood was still obtained from the torcular end. No blood was present near the jugular bulb; the sinus was again packed with four sinus plugs, and mastoid wound dressed. The neck wound was also inspected and appeared to be clean and not infected.

Examination of the throat failed to reveal any evidence of infection. The same day he was given another 1,000 cc. of glucose intravenously and a transfusion of 500 cc. of blood.

The result of the blood culture was as follows: The culture taken on day of admission was sterile. The blood culture taken from the blood of the sinus showed the presence of streptococcus hemolyticus. The same organism was found in the culture obtained from the mastoid curretting and pus found in the mastoid cells. The blood culture taken on July 4 was sterile.

On the evening of July 11, patient had a severe chill which lasted 20 minutes; temperature after the chill rose to 105.6°, pulse 124 and respirations 28. July 12, patient's temperature was normal and he was feeling perfectly well. On July 13, patient was again taken to the operating room for a sinus dressing. All the sinus plugs were removed.

Free blood came from the torcular end. The lower end of the sinus near the jugular bulb was dry. The bulb was probed, no blood or pus obtained. The mastoid wound was temporarily packed and I decided to investigate the neck wound and internal jugular vein. With the aid of the attached black silk ligature around the internal jugular vein, the carotid sheath was then exposed a second time. The internal jugular vein identified. This was found to be bulging and about three times its normal size. The whole length of the vein was carefully dissected with blunt dissection and freed from its surrounding structures as high as possible into the neck.

Iodoform gauze was gently packed underneath and around the vein. The vein was brought forward into the neck. A nick was made into the wall of this vessel, and to the surprise of all, a gush of greenish pus poured out. The solution of the high temperature was now found.

This pus was apparently accumulating very slowly and was causing the elevation in temperature. A very small rubber catheter was inserted into the lumen of the internal jugular vein and was tied to it with a black silk ligature; this was brought out to the neck wound. Through this catheter, the jugular vein and bulb was irrigated daily with a weak Dakins Solution.

The same day the pus was evacuated the temperature dropped to 101°, and gradually came down to normal on the 14th, and remained normal until the patient was discharged.

On July 18, the mastoid wound was inspected again, the sinus plugs removed; there was no blood coming at this time from the torcular end.

The entire mastoid cavity appeared to be healthy. The wound was dressed with iodoform gauze. The neck wound showed very little pus coming from the catheter in the jugular vein.

On July 21, the patient was discharged from the hospital. Reported to my office for dressings till the 24th of August, when both the neck and head wound were entirely healed.

The very interesting complication of this case was the presence of the abscess in that part of the jugular vein between the jugular bulb and the point where the ligature was applied. The abscess undoubtedly caused the marked rise in temperature with no positive blood culture.

It teaches us also the value of having the internal jugular vein ligated with a long suture and having this ligature brought to the neck wound, so that the jugular vein can be examined and investigated whenever the temperature rises and no cause can be found for same.

967 Forest Avenue.

TONSIL TISSUE AS A HEMOSTATIC.

DR. H. H. AMSDEN, Concord, N. H.

The hemostatic properties of tissue juices have long been recognized in medicine. Thromboplastin, prepared from brain, owes its hemostatic properties to this substance. Freshly cut muscle is used to stop hemorrhage in brain operations. J. B. Greene, of Asheville, N. C., has described a method of holding the tonsil in the fossa after enucleation, and this appears to lessen oozing, though, of course, not adequate for bleeding from a vessel of any considerable size.

The results obtained in a case of epistaxis by the use of freshly cut tonsil tissue were so striking that it seems worth reporting, though it is obviously unwise to make sweeping deductions from insufficient premises.

H. O., age 27 years, salesman, personal and family history negative for hemophilia or other blood dyscrasia. He had a submucous resection nine years ago, followed by hemorrhage severe enough to necessitate postnasal packing. He had had chronic tonsillar infection for about three years, with repeated acute exacerbations, some arthritic symptoms and a left chronic maxillary sinusitis. Physical examination was negative; his bleeding time was four minutes and clotting time, seven minutes, so it was thought that his previous postoperative hemorrhage was a surgical accident without significance. June 12, 1933, his tonsils were removed and a radical operation done on his antrum; there was practically no bleeding from the tonsillar fossae, and free but not excessive bleeding from the nose. The inferior turbinate was not resected nor refracted. The intrasinus drain was removed the following day, and the antrum irrigated with no hemorrhage. On the fifth postoperative day he began to bleed from the nose, so profusely that it was impossible to determine the exact source, but it appeared to come from beneath the lower turbinate. This persisted in spite of adrenalin, thromboplastin and salt pork. Notwithstanding his normal blood findings he was given

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thromboplastin, normal horse serum and hemostatic serum in the belief that he might have some peculiar hemorrhagic dyscrasia. Postnasal plug and nasal packing controlled the bleeding for 48 hours, but it recurred immediately on removing the packing. He was given two direct transfusions, and, as a last resort, the external carotid was tied. A postnasal plug inserted at the time of the carotid ligation was removed at the end of 48 hours, and bleeding immediately recurred in apparently undiminished volume.

Having in mind the hemostatic properties of fresh animal tissue, a recently removed tonsil was cut in strips and packed beneath and around the inferior turbinate. Bleeding stopped almost immediately and did not recur again after removal of the tissue three days later.

REFERENCE.

GREENE, J. B.: Tissue Juices in Tonsillectomy. *Annals of Otology, Rhinology and Laryngology*, XXX111, 1924. THE LARYNGOSCOPE, Oct., 1916.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of January 17, 1934.

Symposium: How to Obviate Failures in the Results of Surgery in Oto-Laryngology.

DR. SAMUEL J. KOPETZKY: We have tonight an unusual program, and we have nothing but home talent. We are, I hope, going to tell each other the truth. In arranging this program it was considered that only those who had reached maturity of experience could afford to appear on such a program. I am very happy to announce the cordial cooperation of the leaders of the profession in oto-laryngology in this city for taking part in this program, and I anticipate that both the presentations and their translation into our transactions will afford a lesson that will be of value.

I am pleased to open the program by presenting one of our own members, a man who knows a great deal on the topic he is to present within the limits of time I have set him. I present to you Dr. David H. Jones, who will speak to you, in the opening paper of this Symposium on "How to Obviate Failures in the Results of Surgery in Oto-Laryngology," with particular regard to tonsillectomy.

(a) **How to Obviate Failures in the Results of Tonsillectomy.** Dr. David H. Jones.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(b) **How to Obviate Failures in the Results of Paranasal Sinus Surgery.** Dr. Robert E. Buckley.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(c) **How to Obviate Failures in the Results of Simple Mastoidectomy.** Dr. Marvin F. Jones.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(d) **How to Obviate Failures in the Results of Radical Mastoidectomy.** Dr. John R. Page.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(e) **How to Obviate Failures in the Results of Blood Vessel Surgery.** Dr. Duncan McPherson.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(f) **How to Obviate Failures in the Results of Surgery of Brain Abscess.** Dr. Leo M. Davidoff.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JOHN D. KERNAN: Mr. Chairman, ladies and gentlemen: As I sat here listening to these papers, I thought to myself that these gentlemen had nothing to tell me at all about failures; that I was not learning a thing from them. That sounds pretty bold to say, but the fact of the matter is that I have

made all those mistakes myself, and a few more. I think the operations on nose and throat and ears have been brought to mechanical perfection. I do not see how, if properly performed, there can be any failures from the mechanical point of view. You take Dr. Jones' paper, for instance. It appears to me that that is the most complete piece of work of its kind that has ever been done. I do not know of any such astonishing report of over 50,000 tonsillectomies, all followed up so carefully. Therefore, I am going to turn away from the mechanical part of this problem, and take up other causes of failure. Dr. Buckley and Dr. Marvin Jones in their papers just hinted at some; that is, Dr. Buckley mentioned judgment in selecting cases, and Dr. Jones spoke of allergy as a cause of failure.

As to the matter of judgment, a good deal depends on what you are doing the operation for. If you are taking out tonsils to cure neuritis or a chronic arthritis, no matter how beautiful an operation you do nor how perfect the pillars afterwards, if there is an abscess around a tooth, you will not cure the neuritis nor the arthritis, and your operation will be a failure. In the matter of taking out tonsils, there are a great many failures nowadays from improperly selecting cases. Of course, no one knows "why tonsils," or why they recur after operation. I do not believe even Dr. Jones knows. He may say he does, but I do not believe it. You may take them out ever so carefully, down on the tongue and up in the supratonsillar fossa, you may clean the pillars, and yet they may come back. I have seen tonsils return again and again, even if perfectly taken out. The posterior pharyngeal wall will be covered with lymphoid tissue even if the tonsils do not recur. There is something in such a patient that encourages the growth of lymphoid tissue. Until we know through research the function of tonsils and the cause and constitutional factor in patients which makes lymphoid tissue grow, we will never get 100 per cent successes in tonsillectomies.

Taking up the question of the way people heal, we have all had this experience. We do a submucous resection, for instance, in our usual way. We very carefully clean the nostrils, use douches, pack carefully, and everything goes well during the operation, but that patient has a bad time; the nose swells, fills up with fibrin; it may bleed, and it may form an abscess. You will not know why. Perhaps another patient done the same day heals perfectly; that is, when you take out the packing, the nose is clear, there never will be any swelling, there never will be any fibrin, and it will heal spontaneously and by primary union. Why this difference? You used the same care and the same technique. It may be the constitutional element. There are some people who tell you they heal quickly, and others who tell you they have a hard time healing. The point is, that in our operations we need more study of the constitutional element.

About asthma: Dr. Buckley spoke about curing asthma. Once in a while, we get a glorious result on asthma or hay fever. I remember once, 20 years ago, in my innocence I did a submucous operation for hay fever. I promised the patient that the operation would cure the hay fever, and I believed it then. Well, it did. It cured the hay fever, and she has had none since. I do not know why, but I am in hopes that some day, by proper study of the constitutional factors, we may find out.

About operating on sinuses: there are two schools. There is a man out in Los Angeles, Dr. Eugene Lewis, who maintains he has never seen a successful result from a paranasal sinus operation. He believes in correcting the diet and correcting the constitutional elements. He believes he can charm away nonmalignant tumors by constitutional treatment and diet. I believe it; he is very reliable. Other men believe in operation, and believe that the failures are due to incompleteness of operation. They strip the nose of everything, from tip to nasopharynx. These men report a series of successes. I maintain that the reason they fail in some and succeed in others is that they do not know, and I certainly do not know, what the constitutional element is that makes for success. That is what it all means. We have not yet mastered

that constitutional element. Let me cite an example. You all know what the mortality used to be in operating on diabetics. Now, after proper preparation of his patient, no surgeon is afraid to operate on a diabetic. Another example has been the experience in cases of laryngectomy. The operation of laryngectomy used to be almost 100 per cent fatal. Consider Dr. MacKenty's experience. He went to work to prepare the patient properly; the heart, the lungs, rest, cleaning them out, and so on. All those elements were controlled. He finally won almost 100 per cent success.

Another thing that has to be studied is the physical conformation of the patient. Dr. George Draper has done a great deal of work on that, examining the shape of the patient, the shape of the head, and so on. He says he can determine how one patient will react to disease, and how the other will react to disease, and also how they will each react to operation.

I am going to ask the Chairman something. I once heard him say that the simple mastoidectomy is just about fool-proof. I would like to know whether he still believes that. There is another point I would like to bring up, and I know this will please him. I would like to ask him if he does not think a good way to avoid failures in some simple mastoid operations is to have proper pictures taken of the petrous tip before operation.

DR. CHARLES J. IMPERATORI: No attempt can be made by one in the discussion of such a broad subject to cover the whole field. My remarks shall be confined to two topics.

1. How to obviate failures in paranasal sinus surgery.

To quote from an article written by Dr. William E. Sauer and appearing in the Transactions of the Twenty-seventh Annual Meeting of the American Laryngological, Rhinological and Otological Society, 1921, page 208, it seems to me to about cover, in a general way, the subject under discussion.

End results—at least good end results—are what the patient seeks, the physician, too, for that matter. With unfortunate end results the patient is concerned purely from a personal standpoint, while the physician, apart from his own personal interests, has also the important scientific side to consider. He must determine the causes of any unpleasant outcome of his operative procedures and devise methods of preventing them.

Here the question arises, "What constitutes end results?" Shambaugh lays emphasis on the fact that moisture in the ear or a little discharge after a radical mastoid operation does not mean failure. So we may say of our sinus work, that though there may be some discharge, if the process has been checked and the major symptoms relieved, there is no reason for considering that the operation has failed. Better a slight discharging frontal sinus without other symptoms than an obliterated one; and a greatly reduced maxillary discharge with relief of symptoms, than to pursue the more radical and often disfiguring operation, in order to have an absolute cessation of the secretion.

In general the results of operations on the paranasal sinuses depend upon the following:

1. Character of the pathologic process. 2. Physical condition of the patient. 3. Operative procedure. 4. Skill of the operator. 5. After-treatment.

Conservative surgery should be the rule in the treatment of paranasal sinus disease, and radical surgery reserved for the exceptional case.

Wallace Dean is of the opinion that one of the essential things determining the prognosis of paranasal sinus disease is whether or not the patient is able to seek a proper climate. Paranasal sinus disease is certainly a very different thing in the moist, cold climate of Iowa from that which they have in the high, dry climate of Tucson, Ariz., or Asheville, N. C.

Results obtained: 1. Relief from all symptoms. 2. Persistence of one or more symptoms. a. Suppuration. b. Pain. c. Other symptoms. 3. New symptoms. a. Crusting. b. Neuralgia. c. Paralysis. d. Other symptoms. 4. Complications. a. Meningitis. b. Orbital abscess. c. Optic neuritis. d. Empyema. e. Fistula. f. Anosmia—malpractice suits have been filed. g. Epiphora—anesthesia, double vision. h. Death.

Perhaps nowhere else in surgery does the skill and judgment of the operator have a more direct bearing on the end results. Thorough yet conservative surgery must be followed. The operator who does too much and the operator who does too little will find end results disappointing both to himself and the patient. The timid operator with his repeated tinkering and the bold operator who advances at the expense of undiseased tissue, will both come to grief.

The following conclusions of Mosher and Judd, in an article, "An Analysis of Seven Cases of Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis," appearing in the THE LARYNGOSCOPE, March, 1933, are very timely:

"In osteomyelitis complicating infection of the frontal sinus, edema of the skin and soft tissues of the forehead is the first sign of infection of the medulla of the bone and of the periosteum. The infection of the myeloid tissue of the bone and of the periosteum occur at the same time, and advance together. The edema of the skin of the forehead is a practical guide to the extent of bone to be removed. This has been proved by the microscopical examination of bone specimens removed at operation. In operating, two large triangular skin flaps give the best exposure and the best drainage. The bone removal should begin beyond the edema, generally at or near the hairline, and be carried downward from normal bone to diseased bone. The X-ray does not give positive findings until necrosis occurs, and therefore is not positive until seven to ten days after edema has shown infection of the medulla. When the X-ray finally is positive the infection of the medulla of the bone has extended from an inch to two inches beyond the necrotic area. Radical operation—multiple radical operations if necessary—offer the best chance of success."

2. How to obviate failures in brain surgery.

Gerber begins a chapter on frontal lobe abscess by saying, "The most important factor in brain abscess is the symptomatology."

To quote from F. X. Dercum, "the diagnosis of brain abscess, I need hardly say, is often extremely difficult."

Granted that a diagnosis is arrived at, if the cases are not operated upon, all die, and statistics show of the operated cases, 25 to 50 per cent recover. The following suggestions in the diagnosis of patients having frontal lobe abscess associated with frontal sinus disease, may be of assistance:

Suggested observations on patients having or suspected of having a frontal lobe abscess associated with frontal sinus disease.

1. Determination of the olfactory function.

2. Estimation of the loss of weight.

3. Determination of change of temperament of the patient and existence of a state of euphoria.

4. Relationship of headaches to the side of the lesion—if any abscess is found.

5. Relationhip of the degree of choked disc to the clinical signs of meningitis and the laboratory findings of the spinal fluid.

6. The predetermination whether the patient's condition may be classified as to the stages of the formation of the abscess, during the clinical course of the disease; that is, onset, remission, manifest and terminal stages.

7. At the time of operation on the frontal sinus and in those patients with a suspected frontal lobe abscess and in whom the inner table is found exposed—excessive intracranial pressure should be noted.

8. The proper liaison between the neurologist and oto-laryngologist.

An analysis of five patients in whom brain abscesses were found complicating frontal sinus disease. The following is a summary of the prominent symptoms:

1. Headache, especially at night and localized to the side in which the abscess was found. This should be considered as the cardinal symptom and particularly so in young people with sinus disease or those in whom some nasal operation has recently been done.

2. Mental condition. Euphoric state, that is, a jocose, flippant and discordant emotional reaction of well being that is out of proportion to the apparent seriousness of the patient's illness. Confusions of thought and mild delirium, loquacity, amnesia episodes.

3. Convulsions that were of the Jacksonian type and on the side opposite the lesion.

4. Occasional vomiting.

5. Chemosis and some eye ground changes. Rarely more than one to two diopters of inflammatory process. The engorgement of the retinal veins on the nasal side has been observed. Papilloedema has not been noted. Optic neuritis when the abscess is localized.

6. The abdominal reflexes are disturbed on the side opposite to the lesion. When meningitis ensues, the abdominal reflexes are diminished on both sides. Ataxia and disturbance of station is present when the lesion encroaches on the frontoparietal region. When the lesion encroaches on the postcentral convolution of this area, the disturbance noted is chiefly in disabilities connected with the recognition of size and shape of objects. Also the inability to discriminate between one and two points which are promptly recognized by the normal individual at 3 mm. or less. Other neurological findings such as exaggerated knee jerks, ankle clonus usually found.

7. High temperature toward morning and associated with headaches.

8. Pulse rate usually in relation to the temperature and in no case was it noted to be low.

9. Spinal fluid usually high cell count and no bacteria. Leukocytes usually above 15,000.

In brain abscess occurring in the middle and posterior cranial fossae, in an analysis by Takabatak, who reported from Koerner's Clinic 119 cases of intracranial lesions of otitic origin, 89 had one lesion and 30 had multiple lesions. Of the 89 cases, 42 were perisinous abscess, 16 extradural abscesses, 21 sinus thrombosis, eight meningitis, and two brain abscesses. In the total of 119 cases, there were five cerebral and three cerebellar abscesses.

Heimann collected 435 cases of abscess of the brain, of which 398 were cerebral and 137 cerebellar.

In an article by Frank L. Dennis, of Colorado Springs, "Cerebellar Abscess of Otitic Origin with Report of Case,"—"Judging from the outcome in the cases of so-called stalk abscess, where adhesions between dura and pia have already been formed, the method of Le Maitre or the performance of the operation in two stages several days apart would seem to be more likely to prevent infection of the meninges. If dural adhesions have already formed, the knife is the preferable exploring instrument.

(To be continued in a subsequent issue.)

AMERICAN OTOLOGICAL SOCIETY.

Sixty-Sixth Annual Meeting, May 8 and 9, 1933.

(Continued from a previous issue.)

DISCUSSION.

DR. J. GORDON WILSON: The statements that in otosclerosis the bone changes are confined to the aural labyrinth, that the foci are bilaterally symmetrical and that there is a site of predilection are generally recognized as true and call for no discussion. It is otherwise with the suggestion that otosclerosis is a regression and his theory regarding the origin of the footplate of the stapes.

In support of the former it is suggested that the fissula is a vestigial organ corresponding to a structure found in certain fish. Dr. Guggenheim is not the first to be puzzled by the fissula and sought for an ancestor. Both Dr. Bast and Dr. Anson have been equally puzzled and sought for it in various mammals or for a trace of it in the fetal stages of various mammals. But so far, no trace has been found outside of man. Since no corresponding structure to the fissula ante fenestram can be found in birds or mammals except man, it is a risky procedure to point to a structure in a low vertebrate form as the predecessor, or homologues of the fissula, without explaining what happened to it or why it disappeared during the million-odd years which intervened between the appearance of fish and the advent of man. Attempts to class as homologues, structures which superficially look alike or occupy the same site in fish and man, with no corresponding structure in intermediate vertebrate divisions, has led many a morphologist astray; for instance, insisting that the teeth of the shark and the teeth of man are homologous structures. The chromosome hypothesis to which Dr. Guggenheim refers is, as he knows, a working hypothesis which may or may not be true and cannot be taken as a basis for such a deduction as he makes.

I am not convinced that the footplate of the stapes is derived from the otic capsule. It certainly does not appear to be so in the human fetus. In very early human embryos the primitive cartilage from which the footplate is derived comes from the second branchial arch. In very early embryos just as cartilage is appearing it can be seen lying in the fossa ovale separated all around from the wall of the fossa by a surrounding mass of mesenchymal tissue.

DR. EMIL AMBERG: Imagination is not always a safe guide, but we must sometimes construct an hypothesis in order to work out a particular result. Osteitis fibroas is a lesion that is entirely distinct from otosclerosis in my opinion. The parathyroid glands have a special bearing on calcium metabolism and perhaps on otosclerosis. Brunner and Stein showed a large number of degenerative changes in connection with otosclerosis. The mechanism of the disease is very important. Ballin, of Detroit, is doing excellent work on the parathyroid in this country. Szasz, of Buda Pesth, had published an account of a case of acromegaly with otosclerosis in which hearing was improved by X-ray therapy of the hypophysis. There are two theories on which this work is based. One accepts the influence of radiation on the hypophysis; the other, the Spanish school, the effect on the sympathetic nervous system. Acromegaly bears direct relation to hardness of hearing, but there seems to be a distinction between the effects of acromegaly and those of parathyroidism. In regard

to genetics, Hanhart followed the history of a Swiss family for three hundred years and found that one original couple was responsible for all the cases of deafness that appeared in descendants during that period. Recessant qualities were dormant and intermarriages produced deafened children. Dr. Guggenheim has spoken of changes in the mechanism of hearing. How did they first come about? The sins of the fathers are apparently visited on the children "to the tenth generation." Consanguineous marriages have some influence on recessive qualities. The influence of father and mother may perhaps not cause changes in the next generation, but in the following generations an influence may be felt, such as in color blindness. Prof. Little has shown in his work on mice that the immediately succeeding generation is not affected, but the next one is. Another point is that there seems to be some positive relation between otosclerosis and carcinoma, but a negative relation between otosclerosis and tuberculosis. It is claimed that members of families with otosclerosis are long-lived. The longer a person lives, the more likely he is to become a victim of cancer. That point must also be considered. It is not only in the ear that recessive tendencies are shown. For instance, in incest, the offspring has been shown to suffer from ichthyosis.

DR. ALFRED LEWY: A few years ago, I read a paper before the Triological Society, describing the experimental feeding of calcium fluoride to white mice. It had long been known that fluorine produces changes in bone, and this experiment was to determine whether the bony labyrinth was also affected. It was found in some of the experimental animals that the bone appeared less mature than in the controls. Further study has shown in one specimen deposition of osteoid tissue on one of the crurae of the stapes and on the labyrinthine wall nearby. This evidence of the effect of fluorine is somewhat clouded by the presence of otitis media which occurred in all of our experimental mice and is, of course, very difficult to avoid. However, the experiment offers a suggestion of a line of biochemical approach in the study of otosclerosis.

DR. MOSES H. LURIE: I would like to ask what is the relation of the oval window in the Rajah to the first pharyngeal pouch. Does Dr. Guggenheim refer to the projection from the ductus endolymphaticus of the animals?

DR. STACY GUILD: In regard to Dr. Guggenheim's statement that he has never seen evidence of an inflammatory process in otosclerotic areas, I would say that in the so-called "active" stage the process frequently has a histological appearance which so resembles some features of osteitis that it seems to me entirely possible that much fibrous tissue may be produced by the reaction. I will be glad to how him such specimens.

DR. GUGGENHEIM, closing: Dr. Wilson is right; this paper may even be stamped as pseudoscientific. However, sometimes truth can be arrived at in this way. We cannot be certain that the accessory endolymph duct in the shark and ray is the forerunner of the Fissula ante fenestram, but it makes interesting speculation. The fact that the Fissula ante fenestram is not present in the intervening stages of evolution is most interesting, but it does not prove that the genes may not have been merely inhibited, and therefore capable of later activity. I am not quite clear as to Dr. Wilson's remarks about the origin of the footplate of the stapes. I think it is derived from the capsule and not from the second arch.

DR. WILSON: I examined a six months' fetus and an earlier one, and in the latter the cartilage was not developed; it was in an earlier stage between mesenchyme and cartilage. I think it is derived as the embryologists teach us, but I would like to believe that Dr. Guggenheim is right. It would help us a good deal but we need more evidence.

DR. GUGGENHEIM: The human specimens I have examined do not show details as clearly as the rat specimens. In the latter the footplate is clearly derived from the capsule; the crura from the second arch. I find at four months that although the footplate and Reichert's ring have fused, the two are different structurally. Gradenigo stated in 1879 that the footplate was not derived from Reichert's cartilage. As for the fissula, Bast has the feeling that

it may be vestigial. I know there is a lack of certainty in my statements. The work is not conclusive, but many of the scientific facts of otosclerosis fit in with the theory of regression. Dr. Amberg misunderstood my statement about osteodystrophia fibrosa. In such conditions the entire osseous system may be involved, while in otosclerosis the foci are confined to the labyrinth. Dr. Amberg asked about genetics. I know too little about genetics to discuss the subject but I try to follow Davenport, who tells us that in otosclerosis we have two dominants—one sex-linked. Dr. Amberg said that otosclerosis may skip certain generations, and show up at a later period. This is, of course, quite true. In the present theory genes of regression may be inhibited only, or permanently stilled. We have obtained recently by Caesarian section, a human embryo of two months from a classical case of otosclerosis. We are hoping this specimen will show very early evidence of otosclerosis, at the precartilage stage; that is, certain abnormalities of the differentiated mesenchymal cells. Dr. Lurie asked if the first pharyngeal pouch of the ray is in direct relation to the oval window.

DR. LURIE: The relation should be the same in the human as in the lower animals.

DR. GUGGENHEIM: The first pouch forms the Eustachian tube, etc., but there is no direct relationship between the first pharyngeal pouch and the oval window, except that the oval window abuts upon the lateral expansion of the pouch (tympanum).

DR. LURIE: The endolymphatic duct in the Rajah opens up on the skin just laterally to the spine and fills the membranous labyrinth with sea water. I don't know whether you call that the oval window or whether you mentioned another aperture. That is the first evidence of the formation of the oval window. Is that related to the first pharyngeal pouch?

DR. GUGGENHEIM: The first sign of development of an oval window is in the herring. It is in the capsule wall and is not to be confused with the openings of the endolymphatic duct. Dr. Lewy's experiments with fluorin are most interesting.

Effects of Drugs on Vestibular Reactions. Dr. Ellison L. Ross, Chicago.

In response to the urgent demand for a specific vestibular drug, we have been working on this subject for the past eight years. We have studied only the nystagmic response to rotations followed by sudden stops.

Large doses of drugs representing the pharmacologic groups showed no specific action except, possibly, in the case of strychnine. Medium doses of the same drugs showed no specific action decisively except in the case of strychnine. This drug reduced the reaction about 20 per cent. Spinal anesthesia with procaine increased the nystagmic response to rotation. Cutting the columns of Goll and Burdoch of one side or the cord in the cervical regions increased the vestibular response on the opposite side.

In order to study the different parts of the labyrinth, observations were made on a number of animals in which one internal ear was destroyed. It was found that after one week such a rotation as tended to increase the pressure toward the ampullae produced a nystagmus toward that side, and a rotation tending to decrease the pressure toward the ampullae produced a nystamus toward the opposite side. It was also found that rotation in one direction in the plane of any set of canals was approximately twice as effective as in the opposite direction. Ewald's observation was verified, in that the tendency of the endolymph to move toward the ampullae of the horizontal canals produced a greater reaction than in the opposite direction and that with the vertical canals the opposite was true.

A series of tests were made on animals with but one labyrinth in the hope that a drug would show an action on such animals where it would not be observed on animals with both labyrinths. This is the work I wish to present today.

The table shows the average results obtained before and after the administration of the drug.

This leads us to the following conclusions: 1. The cerebral depressants did decrease the reaction. However, the estimated degree of general depression was as great or greater than the decrease of vestibular reaction. Caffeine did not increase the reaction to any significant degree. 2. Stimulation of the sympathetic nervous system by cocaine and epinephrine had no significant effect. 3. Strychnine, stimulating the spinal cord, did decrease the vestibular reaction approximately 20 per cent.

DISCUSSION.

DR. J. J. MCNALLY: One of the most interesting facts discovered by Dr. Ross is the inhibitory action of the spinal cord on the labyrinth. Strychnine stimulates the spinal cord, which in its turn inhibits the labyrinth and, therefore, strychnine has an inhibitory effect on the labyrinth. This fact may have a very practical bearing. Our interest in a labyrinthine sedative is very great. We have all been accustomed to use bromides. Would Dr. Ross suggest that the real labyrinthine sedative is strychnine? Does he use a cerebral depressant as a labyrinthine sedative? The cerebrum has a definite effect upon labyrinthine function. One cerebral hemisphere has been removed in the rabbit. The nystagmus from caloric irrigation was found to be exaggerated toward the side of the lesion—De Kleyn uses this fact clinically. If Dr. Ross would remove one hemisphere in the dog, and test for this exaggerated effect on nystagmus and then give cerebral depressants, and retest, some interesting effects might be noted. It is difficult to know just what happens in the labyrinth during stimulation with drugs. De Kleyn and Versteegh injected alcohol into the stomach of the rabbit, and then examined for spontaneous nystagmus. It appeared only when the animal was in one definite position. This would suggest that drug stimulation is extremely involved.

DR. DOUGLAS MACFARLAN: I would like to ask Dr. Ross if he has had any experience with the use of quinine.

DR. ROSS, closing: In regard to the use of strychnine in cases of vestibular disturbance, I think it gave very good results. I much prefer it to the effect obtained by depressants. The patients seem to be in a better attitude, and that, in itself, may be a factor as well as the effect on the labyrinth. I give 1/40 to 1/60 of a grain two or three times a day. In regard to decerebration, I have used hemidecerebrated dogs. Two lived one and one-half years. At postmortem it was shown that not more than 4 or 5 per cent of the remaining hemisphere was present. I never could demonstrate a definite effect of hemidecerebration on the nystagmic response to rotation. Observations of others have been made from a different angle. As to the use of quinine, let me say that I used quinine, and it gave a strong depressant action to the vestibular response. I tried to see whether I got degeneration but could see no such effect.

AFTERNOON SESSION, MAY 8.

The Interpretation of Audiograms. Dr. Stacy R. Guild.

The growing tendency on the part of many otologists to make certain diagnoses almost wholly upon the basis of audiograms is, in my opinion, not justified by the facts. The procedure reflects rather the hope which many of us had when modern type audiometers first began to be used; namely, that for each pathologic lesion of the ear there would prove to be a characteristic audiogram. No one believes more firmly than I do that air conduction acuity for tones can be more accurately determined by the careful use of a well calibrated audiometer than by any other means now available. But even the best of audiograms are records of air conduction acuity only. Complete examinations of every available method are just as necessary as ever. The importance of the classical tuning fork tests in otologic diagnosis is not decreased by the use of any audiometer.

The numerous case histories presented by means of lantern slides (audiograms, other functional and clinical data and histologic sections) are arranged in a sequence which emphasizes the differences which the other data may show when the audiograms are similar. The difficulties of differentiating, by means of the functional data, between true otosclerosis and certain other conductive lesions are stressed. Ears whose audiograms indicate that all high tones are poorly or not heard, while the low tones are well heard, have shown the most consistent pathology of any group thus far studied. The essential lesions are in the basal turn of the cochlea, and consist of partial or complete atrophy of portions of the organ of Corti, the nerve fibres in the osseous spiral lamina, the cells of the spiral ganglion, or of all of these elements.

The necessity of adequate "masking" of one ear while testing the other one is not as generally recognized as it should be. Some of the cases presented stress the importance of this point in the technique of clinical examination, for both air and bone conduction.

DISCUSSION.

Dr. Edmund P. Fowler: I think there is no question that we all agree with Dr. Guild that one cannot make a diagnosis on audiometric tests alone; and that to get an accurate picture of both ears one must use a masking tone to see if there is a great difference in the hearing acuity between the two. The great difficulty of masking sound is that when the hearing is greatly lowered the masking sound is inefficient because it cannot be made to enter the ear in sufficient volume. We do not know how much the ear requires for masking, unless it is first used on both ears to test the efficiency of the masking, then used on one ear alone. I think that the Bárány "noise" apparatus makes too much noise in the room; it really masks both ears when used in one. The best way to mask is to produce the masking tone within the auditory meatus, near the drum membrane, by means of an air current, blowing into the external auditory meatus, through a small nozzle. We must, of course, weigh the family history in connection with the otoscopic examination; give due weight to the course of the disease, the flush of the promontory, etc.

In regard to bone conduction, whether by tuning fork or by audiometer, you should test for bone conduction, even if your tests are not exactly accurate. My experience with bone conduction tests is that bone conduction is seldom down more than air conduction. Dr. Guild's charts show the curve down so far that you cannot get bone conduction by forks or by audiometer, because neither produce sufficient loudness in sound. The bone conduction intensity limits range about the 40 to 50 line with the 2A audiometer. Above 4,000 d.v. bone conduction is very inaccurate. The comparison of air conduction with bone conduction by tuning forks is misleading, because as usually obtained, you get no ratio between the two. If, however, you use the method I advocated some years ago, namely, the alternate placement of the fork, first upon the mastoid, and then before the ear, both for two and one-half seconds, the decrement of the fork is the same for bone as for air. It dies down, in other words, in very small steps, but in an even manner for both A.C. and B.C.

Regarding the inflation of ears showing a step-up result because of aeration of the middle ear, I am rather skeptical. In my opinion, if the Eustachian tube is really closed, the drum becomes plastered against the promontory. The majority of otosclerotics have over-inflated middle ears, due to over-treatment. In the Hudson tubes, in normal persons, there is a sense of pressure, usually relieved by swallowing, but some have to blow the nose hard to relieve the sensation and re-establish patency of the Eustachian tube. Often the reason that a tube does not appear patent is that the swallowing is not coordinated with increase in air pressure.

Regarding bone conduction tests with the forks, do not press the fork too hard upon the skull because it causes strong damping of the forks. With reference to cases with perforation of the drum, we have all seen cases with no drum left, but with almost normal hearing. Often an ear with chronic sup-

purative otitis hears better than the other ear wherein the perforation has healed; and after one has done a radical mastoid operation and cured the suppuration, the hearing may be worse. If the suppuration originates from the Eustachian tube it is safe to say that the patient will die of something else, but if it arises in the attic, it is a real danger to the patient. Therefore, we must determine where the chronic suppuration is located. We should try to cure the suppuration in attic and mastoid but even if it is in the latter, it is not always necessary to do a radical operation. As to the hearing after a skin graft, whether we use it or not, the hearing depends on the amount of scar tissue that remains. The main point that Dr. Guild notes is correct: that we cannot make a diagnosis from the audiometer alone. It is obvious that any obstruction in the middle ear has an action depending upon its location. The most important area is the footplate of the stapes. Deafness in otosclerosis is caused not only by ankylosis of the footplate to the margin of the oval window, but frequently by adhesions around the footplate of the stapes as well, or alone.

Dr. Samuel J. Crowe: I think a great amount of information can come from experimental and pathological work, particularly when an effort is made to correlate it with clinical findings. Of course, the experimental results obtained by testing animals with the Wever and Bray method cannot be interpreted literally to explain the defects in hearing observed in patients, and to a less extent this applies to the study of histological section of the human ear. Both of these methods, however, furnish accurate data on which to base an opinion. The entire staff of the Otological Research Laboratory in Baltimore has been engaged during the past three years in an effort to find a rational anatomic and pathologic interpretation of audiometer and tuning fork tests made on patients with the commonest of all hearing defects — a loss or impairment of high tone perception. Dr. Guild has mentioned these studies very briefly, since the main object of his talk today was to direct the attention of the clinical otologist to the fact that an audiometer test alone is entirely inadequate. A careful and detailed clinical history, examination of the tympanic membrane, Eustachian tube, upper air passages, the cranial nerves and the general physical condition is necessary, but above all things a detailed tuning fork test is absolutely essential. The audiometer record supplements but in no way replaces the older, well established methods of examination.

The main object of our research laboratory is to try and correlate experimental findings, audiometer records, tuning fork tests and clinical data on a large group of patients and thus furnish the otologist with anatomic and pathologic facts on which to base the interpretation of his clinical tests.

Dr. Robert Sonnenschein emphasized the fact that laboratory work must check with the clinical by postmortem studies wherever possible; and he called attention to the fact that Bezold did all his splendid work by means of clinical examination with tuning forks and voice tests, together with postmortem findings wherever they were available. Likewise, he stated that audiometers have many advantages, but that they are not indispensable in the matter of making a diagnosis of otological cases. In testing bone conduction he feels that the fork is more reliable than audiometers. For the very highest tones, the monochord is most reliable, both for air and bone conduction. In order to eliminate the objections that are usually raised against the Rinné test, Dr. Sonnenschein and Mr. Minton modified this test a good many years ago, so that the stem of the fork could be used both for air and bone conduction. It is important when speaking of the Rinné test to state not merely that it is negative or positive, but to tell to which degree air conduction is longer than bone conduction, or vice versa. Neumann's electrical noise apparatus is better than the Bárány apparatus as it does not produce as much extraneous noise.

Dr. C. C. Bunch: I quite agree with Dr. Guild in the statement that one cannot make a diagnosis on the basis of the audiogram alone. Complete and thorough clinical examination is always essential. Those of us who are constantly using the audiometer find errors in our technique which often will account for peculiar and incomprehensible results. Simple matters, such as

the proper adjustment of the telephone receiver at the ears, the proper tension of the head band, if one is used, the conditions of noise in the room where tests are made, all play an important part in securing accurate results. These factors are especially important where one is interested in making repeated tests on the same patient in order to determine the progress of deafness, should treatment be undertaken.

DR. LOUIS K. GUGGENHEIM: We seem to forget that the end-organ is not the whole story. In the medulla, at the level of the inferior olives, are the cochlear nerves, anterior cochlearis nuclei, tubercula acustica, etc. From the tuberculum pass the stria acousticae, etc. I would like to ask whether the workers at Johns Hopkins have studied these intracranial structures along with the end-organ, when the audiograms do not "make sense" to see if the difficulty may not be explained by changes in the central structures.

DR. HORACE NEWHART: We must remember that audiograms have now reached the teachers and the laity. The other day I was asked whether an aging teaching should be asked to resign on account of her audiogram. The men here may be taxed wit ha similar question, and I think we should give but one answer in such a case; that is, that the status of a teacher should be based on her record and her performance, and not upon audiogram or tuning fork records.

DR. EMIL AMBERG: It has been my experience to find in some examination of school children, that the ear was obstructed by wax after the audiogram was obtained. Of course, the wax was removed, and another audiometer test requested. This is a little point, but important. I should like to call the attention of the members to an alleged racket that is going on in our part of the country in connection with the aurotone or oratone. Dr. Shambaugh has undertaken to examine the results obtained by this hearing gymnastic machine. The results and his publication in the *Journal of the American Medical Association* should be read carefully. It is distinctly the duty of the medical profession to look into this matter and to advise patients according to the merit or lack of merit of the apparatus. The latter has a wonderful psychological effect, and I can well understand that it has a general sedative effect when our nerves are on edge.

DR. DOUGLAS MACFARLAN: In our tests quantitatively I make use of the tuning fork. We use a technique with a standardized blow,—a swinging pleximeter. Timing the fork gives the best knowledge of the contrast between A.C. and B.C. and is very valuable in making a differential diagnosis. I think the "on and off" method with the tuning fork is the best one for getting the distinction between air and bone conduction. With it you can use one blow and let it die out evenly, and the patient gets the contrast between sound and the absence of sound. This method is important for another reason—the deafened patients are prone to get the impression of tone image with the single tone, if exposed continuously to it.

I am sorry Dr. Guild did not have time to speak about the phonographic audiometer which takes the place of the old spoken voice test. The phonograph gives you an idea of central perception which is a complicated affair, dependent to a large degree upon educational and psychological factors. There is no correlation between single tone hearing and speech hearing. In listening to speech you are listening to a very complex thing.

On all of our records there should be notes as to the "set" of the test, and as to the type of patient. An idea should be given of the sort of individual we are dealing with, whether he is alert or dull, intelligent or illiterate. If it is a child—is he eager, timid, cooperative, resistant, indecisive, etc.? These factors are very important.

DR. EDMUND P. FOWLER: Dr. Guild says that a certain type of curve is associated with ganglionic disturbance, but if we get normal bone conduction I cannot see how we can have much nerve disturbance. Bone conduction is the greatest aid in determining nerve deafness and yet Dr. Guild does not use it. As regards tuning forks, Dr. Sonnenschein misunderstood what I said. The

audiometer as used is more reliable than the tuning fork as used. When you use the fork in the customary way for A.C. and B.C. you might as well use two instruments, one of which gives so many seconds response, and one which gives much less and dies down much faster because of pressure on the head.

DR. STACY R. GUILD, closing: From the amount of discussion it is evident that there are many others who have difficulties in interpreting functional otological examinations. I did not mean to give the impression that I am discouraged, merely that a great deal more work remains to be done. The more complete the clinical examinations, the better are the chances for final success in correlating the several items and the whole picture, with the underlying forms of pathology.

Tactile Sensation as Related to Hearing Testing, and Hearing Impressions Through Nerves Other Than the Eighth. Dr. Douglas Macfarlan, Philadelphia.

The response to the feeling of the vibrations from testing instruments used to produce sound frequently deceives the examiner into thinking that the patient is reporting sound. Many of the profoundly deaf, especially children, have little or no concept of sound. We must then be sure of what they are reporting to us. This has necessitated a more thorough understanding of what is the normal tactile response about the ear.

Vibrations between 128 and 2,000 may be felt if delivered in strong enough intensities. The deafened apparently show a greater tactile acuity than do the normal individuals, for the reason that, with the latter, the hearing of the loud sounds these instruments produce overshadows the first sensations of feeling the vibrations.

A convenient test for making the differentiation between feeling and hearing is one described by the author. A sounding tuning fork is held vertically with one edge facing the external auditory meatus. The patient is directed to report by raising his hand when sensation is noticed. The hand is to be dropped when the sensation is gone. Both the greatest sound and greatest tactile stimulus is coming from the flat of the tyne which is facing the pinna. Tactile sense should first disappear as it is less acute, and fades rapidly with the dying down of the fork. When the patient's hand is dropped, the fork is rotated so that the flat of the tyne faces the meatus. If the hand is again raised, the patient is reported hearing.

Graphs are shown illustrating the relative acuity of touch sense about the ear as compared with other locations in the body. The amount of overlapping of the two senses about the ear is illustrated.

A case is reported whose hearing could be reached by greatly amplified sound. Various pitches of great loudness presented to the ear could be imitated by the patient. Sound delivered by a loud speaker unit, minus the cone, was perceived as sound by the finger tips. The same sound was not sufficiently strong to have been air-borne to the ear. The phenomenon was not bone conduction as the effect was not produced with the sound on the olecronon, but it was exhibited from the ulnar nerve in its groove, a fraction of an inch from the olecronon. The patient could exactly match the pitches perceived in this manner, and could recognize the difference between phonographic speech, instrumental music and singing, sent out through an electrical pickup.

The only explanation ventured is that the crude tactile sense occasionally exhibits the rudiments and perception of sound frequencies which are so well perceived by the special organ of sense, the ear, which latter develops from the crude tactile sense.

(To be continued in a subsequent issue.)

AMERICAN OTOLOGICAL SOCIETY, INC.

The Sixty-seventh Annual Meeting of the American Otological Society, Inc., was held under the presidency of Dr. John R. Page, of New York, at the Claridge Hotel, Atlantic City, April 6 and 7, 1934. The meeting, in the opinion of those present, maintained the high standard of preceding meetings in the character of the papers presented and the discussions which followed. There were present, 49 active members, 4 honorary members and 33 guests. The Society numbers 110 active, 38 senior and 11 honorary members.

Dr. Samuel J. Crowe, Dr. Francis R. Packard, Dr. Thomas J. Harris, Dr. George L. Tobey, Jr., Dr. John R. Page, Dr. Robert Sonnenschein and Dr. Perry Goldsmith were elected members of the Council for 1934-35.

The Council elected from its number as officers for the ensuing year: Dr. Samuel J. Crowe, President; Dr. Francis R. Packard, Vice-President, and Dr. Thomas J. Harris, Secretary-Treasurer.

It was voted that a certificate of the American Board of Otolaryngology shall be essential in the future for membership in the Society, unless by a two-thirds vote of the members present, the Society shall waive this requirement.

The Committee on the Study of Otosclerosis presented an interesting report of its activities during the past year.

The American Board of Otolaryngology reported that since the organization of the Board in 1924, 24 examinations had been held in 20 cities and to date have certified 2,097 otolaryngologists.

The members of the Society are urged to fill out and return to the Secretary the Biographical questionnaire sent them some time ago. To date only 65 questionnaires have been returned. They will be of the greatest value when the history of the Society is written. The Secretary, Dr. Thomas J. Harris, will be glad to supply another blank in case the original has been mislaid.